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EDITORIAL

THE extent to which injury predisposes to tuberculosis has long been the subject of debate. A man develops tuberculosis of the knee and then remembers some slight injury weeks or months previously. Pulmonary tuberculosis starting after a motor accident or any other injury, especially if the thorax was involved, is usually attributed to the injury and an action for damages often results. Drs. Pagel and MacCallum, in the paper which we publish in this number, describe a case where hæmoptysis occurred as a result of reactivation of old foci in the lungs following an injury. The injury does not appear to determine the site of the tuberculous lesion. It was remarkably rare for tuberculosis to follow chest wounds during the war, but in certain cases a penetrating wound of the lung did appear to be an important factor. It is difficult to prove that tuberculosis did not arise from some injury, and in many cases the flimsiest evidence is accepted as proving that it did. If, however, injury does play any part in the production of tuberculosis, it is difficult to understand why even severe injury is followed by the disease in only an infinitesimal percentage of cases. It is known that silicosis is commonly followed by tuberculosis of the lung, and this was taken to show how injury by hard stone could lead to tuberculous activity. Speaking at the opening meeting of the Tuberculosis Association on November 22, Dr. Heffernan explained that silicosis was a special disease due to electro-chemical action and not to mechanical injury by the silica. It is the small particles of silica which cause the damage, large ones are inert. Tuberculosis does not follow injury to the lung if caused by other dusts.

In cases of recognised tuberculosis injury or privation may aggravate

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the disease, but it is extremely rare for activity to start in the injured part. For example, how often does tuberculosis of an ankle follow the spraining or other injury to the ankle, even in the case of a consumptive? It would seem that injury plays a very small part in predisposing to tuberculosis, but further research on this subject is needed, especially as to the effect of injury on old tuberculous foci.

PROBLEMS IN APPLIED MEDICINE

WHY CAN SOME INDIVIDUALS DEVELOP EXTENSIVE TUBERCULOUS LESIONS WITHOUT SERIOUS IMPAIRMENT OF HEALTH?

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The frequent disproportion between the actual extent of structural damage in the lung resulting from its invasion by the bacillus of Koch and the outward clinical manifestations of phthisis is one of the most striking and, at the same time, least understood problems in practical medicine. A real solution of this difficulty might well lead not only to a more intimate understanding of the whole question of tuberculosis, but also to considerable advance in therapy. Since the first recognition of the specific infective nature of tuberculosis, over half a century ago, the earlier and somewhat rigid system of classification has been found to be misleading and has given place to others based on a clearer understanding, supplied by recent studies in immunology, of the behaviour of the disease. It appears necessary, in order to grasp the significance of the question by which this paper is introduced, to insist upon a conception of pulmonary tuberculosis not so much as a disease of the lungs but rather as a general disease in which the local lesion is usually prominent; moreover, this idea must be emphasised not merely in such acute phases as miliary tuberculosis, but equally in those more chronic and relatively restricted forms which provide the majority of cases of consumption.

The development of extensive pulmonary lesions, incommensurate with the apparent state of health of the patient, is seen in a variety of instances; these may, for the purpose of this discussion, be divided into two main groups. In the first must be included those extremely chronic forms of the disease in which the processes of repair tend always to predominate

over those of destruction; although the patients give abundant evidence of gross structural alteration of the lung tissue, and may even continue for years to expectorate bacilli in their sputum, the general health and nutrition remain surprisingly good, and functional activity and efficiency is to all intents and purposes unimpaired. The second group comprises those individuals in whom the inflammatory processes in the lung are of the exudative rather than the productive character, in whom the course of the disease is more rapid in its progress towards an ultimately fatal issue, but in whom the actual spread of a localised lesion, either to the opposite lung or to other and hitherto unaffected portions of the same lung, is unattended by any symptoms of appreciable degree or, indeed, by any external evidence save that furnished by radiology.

Both these types of case must be considered in connection with the question we have asked, and it may assist the reader if they are exemplified by the following brief clinical notes.

CASE 1.—A. W., a married woman aged forty, was found in 1922 to be suffering from phthisis; tubercle bacilli were found in her sputum at that time. She remained in very fair health for about eight years. In the spring of 1930 she had a slight breakdown, following a so-called influenza attack, and began to complain of persistent cough with considerable expectoration, her temperature-chart showing a regular evening rise. She was sent to the seaside at the beginning of August, where she stayed in a comfortable residential bungalow and improved considerably in general health, her temperature settling down to a normal level. She gained a little weight, and the sputum, which had been copious, decreased to about 1 to 2 ounces a day, though tubercle bacilli were still found to be present. In October, 1930, I was asked to see her and to advise as to prognosis and general treatment. When I examined her, her general condition was very fair. She appeared to be rather thin but was not badly nourished, her weight, in ordinary clothes, being 8 stone 12 pounds. Her appetite was good and she complained of no undue fatigue. There was slight morning cough with scanty expectoration. The physical signs in the chest indicated fairly extensive disease of the left lung, the movements of the chest being obviously diminished on this side, dullness and bronchial breathing, with sticky rhonchi, being present over the upper half of the left lung in front and behind. The larynx appeared normal. X-ray examination on the screen showed restricted movement of the left side of the diaphragm, the heart, mediastinum, and trachea being displaced to the left. Radiograms showed appearances indicative of fibrosis and a fairly large cavity in the upper zone, with scattered infiltration in the middle zone of the left lung. The right lung showed compensatory emphysema with some scattered infiltration in the upper and middle zones.

Although there was abundant evidence of extensive involvement of both lungs by disease, which had progressed so far as to result in the formation of a fair-sized cavity on one side, and although the presence of tubercle

bacilli in the sputum showed that some active infiltration was still in progress, her general health was very little disturbed. In view of her age (forty-eight) at the time of examination, and the length of time which had elapsed since the original discovery of the disease (eight years), I felt that her prognosis was undoubtedly favourable, and I saw no reason at the time to suggest any special form of treatment, or indeed any radical change in her mode of living. I have not seen her since that time, but her own doctor, in a recent communication (November, 1935), informs me that she is leading a more or less ordinary life, with a very fair amount of activity—walking, driving, etc.—and that she is free from any distressing symptoms and enjoying very good average health.

Case 2.—M. B., aged forty-seven, an unmarried woman, was sent to me by her doctor with a request to take her into a nursing home for general observation and treatment. She had led a very restricted life at home, her energies being entirely devoted to the care of an invalid mother, who ultimately died of heart failure. She had never been permitted to enjoy any really active life of her own, and on her mother's death she lapsed into an introspective and typically neurasthenic condition, the chief features of which were general debility with loss of weight, coupled with numerous subjective symptoms, mainly referable to her digestive system. Her own doctor had never been able to find any objective evidence of gross organic disease. X-ray examination, after a barium meal, revealed only a general visceroptosis, and the present condition was not unnaturally attributed to the climacteric, aggravated by her general environment and by the psychological disturbance occasioned by her bereavement and consequent loneliness.

Physical examination showed little beyond a generally poor physique. Among all her numerous symptoms, cough and expectoration were conspicuous by their absence. On examination of the chest there was dullness, with bronchial breathing in both supraspinous fossæ, no adventitious sounds being audible. X-ray examination revealed fairly extensive fibrosis of the upper zones of both lungs with scattered calcified foci in these areas, and calcified hilar glands. A moderate-sized cavity with a well-defined fibrous wall was seen in the right upper lobe.

She was kept for some weeks in the nursing home on a Weir-Mitchell régime. General massage was given daily by a skilled masseur, and her metabolism was stimulated by injections of insulin (5 units twice daily at first, increasing later to 10 units twice daily), appropriate quantities of glucose and other carbohydrate being given by mouth. At the end of four weeks she was sent to a convalescent home in the country, whence, after a further rest-period of about six weeks, she returned home. When I last heard from her (October, 1935) she was feeling much better, had put on half a stone in weight, and was leading a more or less normal life and interesting herself in various forms of social work.

The two foregoing cases are good examples of the maintenance of very fair general health and working efficiency in individuals who, despite the presence of extensive tuberculous lesions in the lungs, were more or less free from obvious symptoms of disease. It would be easy to multiply in-

stances of such patients. In regard to the more acute and unfavourable type of disease the following example will suffice.

CASE 3.—E. R., a girl aged twenty, a worker in a factory, developed a right-sided pleural effusion in the early spring of 1933. She was kept in bed for about nine weeks, during which period the fluid gradually absorbed and the temperature, which at first was high, settled to a normal level. She was then allowed up, and after a suitable interval was sent to the South Coast for convalescence. I was asked to see her in May of the same year to advise as to her fitness for return to work. She then weighed 8 stone 10 pounds in ordinary clothing, was free from all symptoms, looked well, and declared that she felt well and was anxious to return to her work. The only abnormal physical sign found on examination of the chest was a slight diminution in the respiratory murmur at the right base as compared with the left side, the vocal fremitus and resonance being normal; no appreciable alteration of resonance to percussion was detected.

I was on the point of saying that I thought she could reasonably return to work in about a week, but before doing so suggested that it would be wiser to have an X-ray examination of the chest. Screen examination showed restricted movement of the left side of the diaphragm. No residual effusion was apparent on the right side, though there was some loss of translucency at the base. Radiographs showed fibrosis and infiltration of the upper and middle zones of the right lung, and extensive infiltration of the upper and middle zones of the left lung. The opacity in the left upper zone was denser and more homogeneous than that on the right side, and suggested a recent exudative lesion of the lung. In view of these appearances she was admitted to hospital for further observation, when she was found to have a slight evening pyrexia. Within a few days of admission, fine râles were heard over the upper and middle zones on the left side, and a specimen of sputum was ultimately obtained which was found to contain tubercle bacilli.

After a few weeks in hospital the temperature again became normal, and she was transferred to a sanatorium, where she made some improvement on routine treatment; after a few months she returned home, the prognosis, in view of her age and the character of the lesions as suggested by X-ray appearances, being regarded as very unfavourable. The last information I have of this patient is in a communication from her own doctor (March, 1935), in which he says: " She has been confined to bed for the past month, the night-temperature being 100° F. or thereabouts. She had kept very well until a month ago, when she developed a severe cold. I am afraid she is now starting to go downhill; there is loss of weight, pyrexia, and a definite extension of the tuberculous lesions."

It will appear from the preceding notes and observations that we are dealing with two main types of case, which differ a good deal in their general characteristics and in their morbid anatomy. One is the chronic fibro-caseous tuberculosis of the lung, seen mostly in older people in whom there has at no time been any very rapid or uncontrolled spread of the disease,

the activity of which has been limited by the barrier of fibrous tissue which tends to localise the inflammatory process. The other is the more rapid exudative tuberculosis which characterises the so-called "young adult type," and in which the fibrous barrier is less in evidence. In both of these, however, the same essential phenomenon of relative immunity is apparent, more permanent in the former class, transient and less permanent in the latter. Both types of case must be studied if we are to arrive nearer to an understanding of the question why one individual remains in comparatively good health, despite the presence of extensive lesions, while another goes steadily and even rapidly downhill, although everything has been done to render the patient's environment as favourable as possible.

Although, in the present state of our knowledge, it is impossible to speak with any certainty of the conditions which determine the spread or activity of tuberculous disease, I cannot help feeling that the essential difference between the curable and the incurable case of phthisis lies in the soil rather than in the seed, and that the answer to our question will come from further investigation along biochemical lines. The comparative immunity enjoyed by phthisical women during pregnancy is a phenomenon familiar enough to those who have observed large numbers of cases; no less striking is the acute exacerbation of disease which so frequently follows labour, especially if the latter be unduly prolonged. Apart from the increase of auto-inoculation, which may be assumed to occur as the natural corollary of strenuous activity of the uterine muscle, it would seem likely that some additional factor must be present—*e.g.*, the removal from the circulation of some substance which has been supplied during the previous months in consequence of the altered chemistry of the body which is an accompaniment of pregnancy. That the exacerbation of activity after labour is associated with a marked disturbance of the general endocrine balance, occurring in the weeks immediately succeeding the emptying of the uterus at term, is a not unreasonable hypothesis, and one which gives food for thought as to the possible lines on which research may in the future be conducted into the difficult problem of relapse in tuberculosis.

The latter has been discussed at some length by Wingfield, whose admirable summary, in the last issue of this JOURNAL, of the different forms of relapse has stimulated me to speculation on these lines, and to hope that still further research may be conducted into the relation between tuberculo-allergy and immunity, a better understanding of which is, I feel sure, essential to advance in treatment. That allergy and immunity are two different things is, I think, generally understood. That a high degree of hypersensitivity is often associated with a liability to the occurrence of the "new lesion relapses," described by Wingfield, is, I believe, a fact, and

attempts have already been made to desensitise such patients, in the stages in which they are clinically healthy, by carefully controlled administration of tuberculin B.E.

The hypothesis that the varying results to the individual of a tuberculous lesion of the lung depend upon inherent differences in the bodily make-up of patients seems to me to gather support from the work which has been done by Lyle Cummins, Houghton, and others, on blood assessment in tuberculosis. It has been shown that patients with a low blood index, who improved on routine treatment in a sanatorium up to the point at which they were afebrile, increased in weight, and were more or less free from symptoms, nevertheless relapsed within a comparatively short time of leaving the institution, and usually died early. Others, whose blood index was more favourable, ultimately did much better, and tended to survive. Trail has had a similar experience in connection with the simpler method of blood examination by taking the sedimentation-rate alone.

The question of the presence or absence of constitutional symptoms in a patient who has an extensive pulmonary lesion is a complicated one which may well depend on several factors. It has been suggested by more than one observer that the actual products of activity of the bacillus itself are multiple, one being responsible for tissue destruction, and another for general constitutional disturbance which results in the more active toxæmia that is directly responsible for the patient's manifest feeling of ill-health. Another factor which has to be considered is the activity of non-tuberculous pyogenic organisms as well as the conditions which govern the incidence of secondary infection. It is hardly possible, at present, to speak with decision on these important points, or to say just what is the relation of the various factors concerned with the local manifestations of the disease to those inherent conditions which constitute the peculiar resistance-ratio of the individual. I am inclined to think, from general pathological considerations, that the latter aspect of the problem is, perhaps, the more important; it certainly appears to be the more neglected. The occasional development of a rapid and intractable form of the disease in several members of a family successively would seem to support the belief that some inborn peculiarity of metabolism is mainly responsible. That such a peculiarity may be inherited is an idea which is at least consonant with accepted laws of heredity.

Such are the general considerations which must occupy our minds in the attempt to deal with the question which forms the subject of this paper, the answer to which can as yet hardly be more than speculative, but which, I feel, may possibly be supplied in the future by greater attention to the humoral pathology of the individual, on the lines which I have endeavoured to indicate.

GENERAL ARTICLES

THE TREATMENT OF PULMONARY TUBERCULOSIS BY PHRENIC EVULSION

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THE material of this paper consists of 316 cases of phrenic evulsion operated on between 1930 and 1934, nearly all of whom were treated at the Forster Green Hospital. Excluding 2 non-tubercular and 9 abacillary cases, 3 cases without positive functional result, and 2 cases where the indication for operation was merely symptomatic, there remain 300 patients. Tubercle bacilli were demonstrated in the sputum of every one of these 300 patients.

The type of operation done on all the cases has been a Felix exairesis, but at the same time accessory nerves have been looked for arising from the fifth cervical nerve, and when found have been divided. Any nerve arising from the upper roots of the plexus and running inwards across the scalenus anticus muscle has been regarded as a possible accessory. We have not done any temporary phrenic crushing.

Except in a few cases at the beginning of our phrenic operations where a moderately long incision was made and a wide surgical exposure done, the phrenic has been approached through a short transverse incision $\frac{3}{4}$ inch to 1 inch long two fingers' breadth above the clavicle, the centre of the incision being situated either directly over the scalenus anticus when palpable or directly above the mid-point of the clavicle. It is situated immediately behind the posterior border of the sterno-mastoid muscle. The line of incision, skin and subcutaneous tissue is infiltrated with novocain (8 to 10 c.c. of 1 per cent.). It has not been found necessary to infiltrate the deeper tissues, fascia, fat, etc. After incising the skin, blunt dissection only

(dissection with fine artery forceps) is used to expose the area overlying the scalenus anticus muscle and its fascia under which the nerve is situated. When found the nerve is tested by lightly pinching with dissecting forceps, which produces a diaphragmatic contraction shown by a quick jerk or "kick" in an instrument placed on the epigastric region of the abdomen. It is picked up with a sharp curved hook, isolated, infiltrated proximally with novocain, divided and its distal end rolled up on a pair of artery forceps. As much as possible of the nerve is removed. In only one or two cases has it been necessary to ligature a vessel.

Anatomically the easy nerve to find is the one running obliquely across the anterior surface of the scalenus at the level of the incision; if the nerve is not seen in this position we have found that it is either the "high" nerve which at the level of the incision has already come to lie on the medial aspect of the scalenus, or the "low" nerve which comes off the fifth cervical nerve and is found at the lateral border of the scalenus. In no case have we found a nerve in the substance of the muscle.

There have been no operative or post-operative complications. No hæmatoma formation or wound infection has occurred. Twice a Horner's syndrome has been noticed, but this has only been temporary, clearing up completely in a few hours. It has been ascribed to the local anæsthetic or to tearing of sympathetic connections to phrenic in doing the exairesis, and so by trauma temporarily blocking the sympathetic.

Results with Reference to Rise of Diaphragm.

Three cases showed no rise of diaphragm. In the first 2½ inches of nerve were removed. Paradoxical movement was observed, but no rise. In the second 4 inches of nerve were removed and the diaphragm on screening showed a normal movement. In the third 3½ inches were removed, but no rise of diaphragm occurred. There is no note of diaphragmatic movement in this case.

An attempt was made to correlate the amount of rise of the diaphragm with the length of nerve removed in 112 cases, but no significant difference was found.

There were several cases which showed a rise of the diaphragm, but with slight normal movement. These we have regarded as successful from the operative point of view.

In 8 cases the lung was so opaque that it was not possible to observe the position and movement of the diaphragm. In all the other cases the result of the operation was verified by X-ray photography or fluoroscopic examination, usually by both these methods.

Up to the present we have regarded artificial pneumothorax as the operation of choice when collapse is indicated, and therefore most of our cases of phrenic evulsion followed a successful or an attempted artificial pneumothorax; 28 patients had a phrenic evulsion in preference to artificial pneumothorax (one of whom had a successful artificial pneumothorax later). The reasons for not trying an artificial pneumothorax in these 28 patients were as follows:

Definite history of pleural effusion	12
Emphysema	9
Very chronic disease	6
Basal lesion (artificial pneumothorax later)	1
Total	28

In the rest of this paper this group is combined with the cases where artificial pneumothorax was tried without success. The 300 cases are divided as follows with regard to artificial pneumothorax:

	Associated Pneumothorax.			
	Satisfactory Collapse.	*No Satisfactory Collapse.*	*No Pneumothorax.*	*Total.*
Discharged, 1930-1933	55	43	63	161
Discharged, 1934	31	22	52	105
In hospital, December 31, 1934	15	12	7	34
Total	101	77	122	300

The considerations which have inclined us to resort to artificial pneumothorax before trying phrenic evulsion are numerous, but perhaps the most weighty one is the danger of delay when artificial pneumothorax is clearly indicated, for every month which elapses increases the risk of pleural adhesions. If one decides to perform phrenic evulsion first, one must wait several months to judge the effect, and by this time adhesions will probably be more numerous and more formidable. On the other hand, one can always rely on phrenic evulsion as a second line of attack, and experience has shown us that phrenic evulsion may be very successful, even when the pleural surfaces adhere densely.

Phrenic Evulsion in Conjunction with Artificial Pneumothorax.

In 178 of the 300 cases there was a more or less successful artificial pneumothorax. However, in 66 patients the lung was already satisfactorily collapsed before phrenic evulsion was done. In 25 patients phrenic evulsion was performed after an unsuccessful artificial pneumothorax had been discontinued. There remain 87 patients to be considered who had an

unsuccessful artificial pneumothorax (the chief criterion of which was an uncollapsed cavity) at the time phrenic evulsion was performed. A temporary improvement in the collapse followed phrenic evulsion in almost every case, and collapse became satisfactory in 35 patients (3 of whom had adhesions cut as an additional measure). The remaining 52 patients did not attain a satisfactory collapse, although usually cavities appeared smaller after the operation.

In successful and unsuccessful cases alike there has been a marked tendency for the artificial pneumothorax cavity to obliterate after phrenic evulsion. We do not agree with the opinion that refill intervals may be lengthened after phrenic evulsion. It is necessary to keep the intervals short in order to prevent obliteration of the air space. Of course, it is useless to employ positive pressures after phrenic evulsion, which only result in pushing down the paralysed diaphragm.

There are two special groups of artificial pneumothorax cases in which phrenic evulsion is urgently indicated. The first indication is a thin-walled cavity held out by adhesions. The prompt performance of phrenic evulsion in these patients tends to prevent rupture of the lung, and this grave complication of artificial pneumothorax has not been seen for two and a half years among our patients. The second indication is hæmorrhage from a partially collapsed lung, which was successfully controlled by phrenic evulsion among four of our cases.

As a method of dealing with pleural adhesions phrenic evulsion is uncertain, and internal pneumolysis is the method of choice. There are, however, contra-indications to a direct attack on pleural adhesions in certain cases which may not debar the patient from phrenic evulsion. In such cases phrenic evulsion may bring about a temporary improvement, and the patient's condition may permit of thoracoscopy and adhesion-cutting some months later. When only one or two thin adhesions appear on the X-ray film, phrenic evulsion is usually followed by a marked improvement in the collapse. When the adhesions are more numerous or appear thicker than $\frac{1}{4}$ inch, the operation is usually ineffective: 33 cases had adhesions over one zone only—of these, 21 had a satisfactory collapse after phrenic evulsion; 40 cases had adhesions over two zones—of whom 13 had a satisfactory collapse after phrenic evulsion, 14 had adhesions over all three zones, and of these 1 had a satisfactory collapse after phrenic evulsion.

Judging by X-ray appearances, one-third to a half of the unsuccessful cases would have been suitable for internal pneumolysis.

Better results have been obtained by combining artificial pneumothorax and phrenic evulsion by Dr. P. Slavin,[1] who succeeded in causing the

disappearance of 18 out of 28 uncollapsed cavities when he supplemented artificial pneumothorax by a phrenic evulsion (64 per cent.).

At the Cheshire Joint Sanatorium Russell[2] found that of 32 uncollapsed upper lobe cavities in artificial pneumothorax cases, 20 had collapse of the cavity after phrenic evulsion (63 per cent.).

One advantage of internal pneumolysis over phrenic evulsion is that it leaves one free to perform phrenic evulsion on the other side at a later stage, if necessary. This group of cases should be suitable for a temporary phrenic interruption, which has not been practised in any of our series.

Analysis of 122 Cases of Phrenic Evulsion where there was no Associated Artificial Pneumothorax.

An attempt has been made to assess the effect of phrenic evulsion on the diseased and healthy lung tissue.

In 26 cases there was no X-ray photograph after phrenic evulsion or the lung was densely opaque. Of the other 96 cases, 35 had evidence of disease fairly equally throughout all areas of the lung, and it was not possible to study the selective effect of the operation in this group. Many of them, however, showed improvement after the operation. Of the other 61 cases, no less than 35 (57 per cent.) showed a well-marked contraction of the diseased area, without any marked diminution in the extent of the healthy lung tissue—*i.e.*, selective contraction.

In 10 patients (17 per cent.) the reverse process occurred—*i.e.*, the healthy lung tissue contracted and the diseased area increased or remained unchanged in size (contra-selective contraction). In the other 16 cases (26 per cent.) the result was indefinite—*i.e.*, the contraction of lung tissue was equal in healthy and diseased areas.

A closer examination of the 10 patients who showed a " contra-selective " collapse after phrenic evulsion reveals the fact that only one of the 10 would have been suitable for thoracoplasty. This patient, who has been lost sight of since discharge from hospital, was advised to return for re-examination with a view to thoracoplasty, if necessary. The 9 other patients constituted a very unfavourable clinical group ; 1 is dead, and most of the others are in poor or indifferent health, but there is no reason to believe that phrenic evulsion had an unfavourable influence on the course of their disease. We have observed much less frequently than Dr. Gravesen[5] the contra-selective contraction of lung tissue after phrenic evulsion, and it appears to us that this unfavourable development occurs chiefly when there is no natural tendency for the lung to heal by contraction.

Of the 35 patients who showed a contraction of the diseased area after

phrenic evulsion, 25 had disease of the upper half of the lung, 10 had disease of the lower half. The 10 patients who responded unfavourably to the operation all had disease of the upper half of the lung. These figures do not support the view that phrenic evulsion is equally valuable in apical and basal lesions. Undoubtedly in our series the basal cases have done better as regards selective contraction of lung tissue.

Of the 16 cases where the effect of operation was "indefinite," 15 had disease of the upper half, 1 had disease of the lower half.

Effect of Phrenic Evulsion on Cavity.

This has been studied in the 122 cases referred to above and also in the group of 25 cases in which phrenic evulsion was done *after* an unsuccessful artificial pneumothorax had been stopped.

Cavities studied	88
No cavity or lung opaque	31
No X-ray control after phrenic evulsion*	25
Cavity healed before phrenic evulsion	3
Total	147

Twenty-two of the 88 cases had multiple cavities and 66 had a single cavity.

	Healed.	*Improved.*	*I.S.Q.*	*Larger.*	*Total.*
Single cavity	22 (33 per cent.)	25	11	8	66
Multiple cavities	1 (5 per cent.)	9	7	5	22

Analysis of the Effect of Phrenic Evulsion on Sixty-six Single Cavities.

	Healed.	*Improved.*	*I.S.Q.*	*Worse.*	*Total.*
Small cavity—*i.e.*, less then 2 inches diameter	21	13	8	7	49
Large cavity—*i.e.*, 2 inches or more in diameter	1	12	3	1	17
Cavity "early type"	21	13	4	4	42
Cavity "late type"	1	12	7	4	24
Cavity in upper zone	11	14	8	3	36
Cavity in middle or lower zone	11	11	3	5	30

The X-ray control after operation varied from one to forty-five months, but the great majority had an X-ray picture twelve months or more after operation. Where more than one post-operative picture was available the most recent one was used for the above tables.

Nehil and Alexander,[3] using phrenic evulsion as an independent measure, found 38 per cent. of cavities closed after operation in an analysis of 179 patients. Naturally, the smaller cavities closed much more frequently than the larger ones.

* Fluoroscopic control only.

Wirth and Jaski[4] have studied the late effect of phrenic evulsion on "tertiary" cavities; 109 "tertiary" cavity cases showed the following result one to five years after operation:

	Per Cent.
Cavity healed	27
Cavity improved	13
Cavity unchanged	20
Cavity worse	40

The results of treatment are as follows in the 161 patients discharged from Forster Green Hospital before December 31, 1933. An attempt has been made to trace all these patients in 1935.

RESULT OF PHRENIC EVULSION, 1930-1933, AS KNOWN IN FEBRUARY, 1935.

	Alive.	*Dead.*	*Untraced.*	*Total.*
A.P. (S.C.). This includes all cases with a satisfactory collapse before or after phrenic evulsion	50	5	—	55
A.P. (N.S.C.). This includes all cases with phrenic evulsion before or after the end of an unsuccessful artificial pneumothorax	25	18	—	43
A.P. (N.C.). This includes all cases where artificial pneumothorax was tried without success or not tried at all	46	13	4	63
Total	121	36	4	161

An attempt has been made to estimate the effect of phrenic evulsion in unsuccessful artificial pneumothorax cases by comparing the results with those obtained in the years before phrenic evulsion was introduced as a method of treatment.

The table below shows the results at the end of the year after the year of discharge from the hospital—*i.e.*, at an average period of eighteen months from the time of discharge:

COMPARISON BETWEEN GROUPS OF UNSUCCESSFUL ARTIFICIAL PNEUMOTHORAX CASES WITH AND WITHOUT PHRENIC EVULSION.

	No Collapse and no Satisfactory Collapse Cases (Phrenic Evulsion).			*No Collapse and no Satisfactory Collapse Cases (No Phrenic Evulsion).*		
	Mod.	*Adv.*	*Total.*	*Mod.*	*Adv.*	*Total.*
Alive	20	66	86	12	29	41
Dead	2	13	15	4	46	50
Untraced	2	3	5	—	—	—
Total	24	82	106	16	75	91

These figures illustrate the value of phrenic evulsion in advanced pulmonary tuberculosis.

The operation puts so little strain on the patient that it is ethical to try it in the most unfavourable types of case, if the disease is mainly unilateral.

Most of our cases were clearly unsuitable for thoracoplasty, and of those which were suitable for thoracoplasty many did so well after phrenic evulsion that the larger operation has not been considered. In a small group of patients with unilateral disease and a cavity of 2 inches diameter or more thoracoplasty is probably the operation of choice (if the patient is willing), and in such cases a permanent phrenic evulsion may be disadvantageous.

Conclusions.

1. Phrenic evulsion has been performed in 316 consecutive cases without any serious complication or ill effect.

2. The operation has been successful in paralysing the diaphragm in approximately 99 per cent. of the cases.

3. The results of phrenic evulsion have been very satisfactory at the end of artificial pneumothorax in a group of cases where it is not advisable to allow the lung to re-expand fully.

4. When a cavity remained uncollapsed with artificial pneumothorax, phrenic evulsion has been successful in effecting collapse in 37 per cent. of our cases.

5. Phrenic evulsion as an independent measure may help to bring about the arrest of pulmonary tuberculosis and the closure of cavities.

6. The usual result of phrenic evulsion is to allow the diseased part of the lung tissue to contract, but occasionally the reverse occurs.

7. Phrenic evulsion is rather less successful in lesions situated in the upper zone than in mid-zone or basal lesions.

8. Forty-three per cent. of cavities of less than 2 inches diameter closed after phrenic evulsion.

9. Of 22 cases of multiple cavity, only 1 showed healing of both the cavities after phrenic evulsion.

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SOME OBSERVATIONS ON THE PLOMBAGE OPERATION IN PULMONARY TUBERCULOSIS

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In my article on " The Operation of Plombage in Pulmonary Tuberculosis " (*Tubercle*, December, 1933, pp. 97-109) I explained the nature and purpose of this method of intervention. Most of all I emphasised the importance of a clear, though narrowly defined indication, and the bearing in mind of certain principles of collapse-therapy, in order to attain good and lasting results. In pulmonary tuberculosis the chief object, apart from establishing a good general condition, must be to abolish cavities and expectoration. For in the study of pulmonary tuberculosis we find, over and over again, cases in which progressive trouble can suddenly recommence from small residual cavities, so that what may at first appear to be a satisfactory result becomes unsatisfactory in course of time because the cavity was not primarily closed, or because this did not occur within a few months after operation.

I have been led, by the result of repeated personal experience combined with impressions received from other sources, to enlarge on certain aspects which appear to me important in the cases of a few specially selected medical histories. I am deliberately choosing cases from among my own patients in which there was a partial success, which, for various reasons, was not maintained. I shall therefore discuss in some detail certain failures such as will be experienced by any specialist who is familiar with this method of treatment.

Case 1.—The patient was born in 1893, and in 1912 developed aortic disease as a result of rheumatism. In 1930 bilateral apical tuberculosis was diagnosed. Nine months of a conservative cure (gold-therapy) improved general condition and sedimentation of red blood-corpuscles, still a minimum quantity of 10 c.c. of sputum was daily secreted. In consideration of the double-sided character of the disease, and with regard to the defective heart, a bi-lateral plombage was recognised as the only possible means of collapse, since an attempt at pneumothorax failed in both lungs, in this instance luckily for the patient. On October 15, 1931, the first operation was performed on the right cavity. It was

sufficiently loosened at the front side and back. It was joined to the base by adhesions of a leathery and bony consistency in a paravertebral direction, about two fingers obliquely from the top. Consequently one had to be satisfied with a partial apicolysis. About 150 g. of paraffin were inserted. Post-operation progress of no importance. On December 15, 1931, the cavity in the left apex was loosened in the customary way and the extra-pleural hollow was filled in with about 100 g. of paraffin. The whole apex allowed itself to be loosened and the collapsed cavity could be felt in the plombage-bed. In both cases the temperature reached 100·4°. On January 16, 1932, a third operation was performed in an attempt to collapse the remaining right cavity. But this time the operation took place from the front after resection of the second rib. It was possible to complete the loosening of the cavity medially, but at the level of the top of the cavity the loosening finger met the obstacle which had already proved insurmountable in the first operation. Into the extrapleural cavity formed, which lay in the front and medially, about 80 g. of paraffin were carefully inserted. Expectoration decreased to 3 c.c., which was discharged in the morning. On May 1, 1932, in spite of positive sputum, the patient returned to active life and worked in good condition. An observation three months later showed a slightly increased sedimentation of the red blood-corpuscles and sputum to the amount of 5 c.c., which was still positive; it originated from the apical cavity on the right side, which had not yet shrunk.

The good general condition of the patient, with complete fitness for work, was maintained for two years, in spite of residual cavities. In the spring of 1934 abuse of alcohol and lack of discipline led to serious failure of compensation of the heart. In the summer the cavity area, which had hitherto been quiescent, again became active, showing itself in a decided increase of expectoration and in an enlargement of the cavity. The patient had to go into a sanatorium again, where he is still in bed, with a serious outlook for the future.

The plombage on the left lung was successful. It was possible to collapse the cavity right away. In the case of the large apical cavity in the right lung, the loosening medially could not be completed, and a second operation from the front was also of no use. I nevertheless recommended the patient to resume his work, which he was able to carry on for two years, leading at first a strictly regular life and suffering no injurious effects. Periodical control showed that the general condition was good, so that if he had continued to lead a temperate life the good results should have lasted much longer. Moreover, the extensive dissemination through the blood stream remained permanently quiescent, so that it must have been only the lack of discipline that caused the fatal relapse, which manifested itself, not only in the enlargement of the remaining cavity but also in a fresh diffuse infiltration at the left lower edge of the plombage. It is impossible as yet to foresee how and to what extent the serious disorder of the heart will determine the man's fate.

Case 2.—This patient had severe progressive tuberculosis with two cavities in the right upper lobe and one in the left. Ten months' conservative cure without success. Hence pneumothorax on the right. Over-

taxing of the left side on account of a too strong collapse of the right lower lobe, which was potentially capable of functioning. In two sittings adhesions at the right apex were cauterised and the cavities collapsed. A small exudate reaction led to a total adhesion of the right lower lobe with the thorax-wall, and since the cavity-collapse lasted sufficiently long, an oleo-thorax was performed. It was borne without a reaction. The left lung remained stationary. The cavity, however, still produced expectoration; hence a pneumothorax on the left side a year later. The cavity was suspended from its apex, consequently three months later the pneumothorax refills were stopped, so that after a lapse of a further three months a left plombage operation could be performed. Three weeks after the operation the patient was free from expectoration for the first time in three years. The operation was performed in January, 1932, and for more than a year her condition was satisfactory and stationary. No bacilli to be found in the very small amount of sputum secreted. The patient was able to undertake light duties. Family circumstances brought disturbing times for her, and she gradually ceased to spare herself, and disregarded all warnings. Attacks of fever with positive sputum ensued, and spreading disease could be diagnosed, so that in a few months the young lady succumbed to a serious progressive phthisis with cavities in both lungs.

In spite of the patient's grave condition, the apical cavities were operated on as soon as the general tuberculosis had become stationary. While the right lung was being treated by collapse-therapy the left side showed no activity, so that it was possible to follow up the first operation by a plombage here as well, which collapsed the apical cavity and resulted in the patient being practically free from expectoration for over a year. It must be specially emphasised that during the application of these various, by no means simple modes of intervention the other foci showed no activity of any kind. From the beginning the prognosis for the general tuberculosis was doubtful, as the apical cavities represented only a part of the disease, and the patient's delicate constitution called for great care, both at that time and afterwards.

Case 3.—A lady of thirty years of age, with a large apical cavity on the left side, and a number of spots on both lungs, especially the right. Had been ill several years. A pneumothorax not possible. A conservative cure for three months proved of no use. In January, 1933, a plombage operation was performed. The apicolysis was extended too far laterally, so that the cavity sank medially. It diminished considerably, but four months after the operation it had to be helped to a complete closing-up by means of a temporary paralysing of the diaphragm. It closed up rapidly, and the patient's condition was satisfactory after this. In fact, she remained negative for some time, but in the spring of 1934 she started fresh trouble in the shape of a new infiltration below the plombage with a new cavity about the size of a walnut, which refused to yield to strict treatment.

It is very unlikely that a permanent paralysis of the diaphragm would cause the cavity to shrink, as the infiltration developed actually during the temporary paralysis, and the left diaphragm recovered its flexibility only in the late summer of 1934. On the other hand, it must not be forgotten that there was no prospect of curing this case by mere

exairesis without a plombage, and a plastic was also out of the question owing to the extensive contralateral disease. Nevertheless the question remains, whether or not a big plombage operation in the first place, which would also have given considerable relief to the tuberculous infiltrated area lying beneath the original cavity, might not have prevented the new cavity from forming.

CASE 4.—The patient was a woman of twenty-eight. At first no notice taken of illness; in December, 1931, an X-ray photograph showed a preponderantly right-sided upper-lobe cavity-tuberculosis. Pneumothorax attempt unsuccessful. Situated just beneath the large apical cavity was a portion of lung with small cavities, which was too extended to allow a proper primary plombage. Since the state of the lung remained unchanged in spite of a six-months' rest cure, the diaphragm was first of all paralysed on the right, in the hope of producing a considerable shrinking of the tissue surrounding the cavity, especially the lower portion. Hence the cavity was plombaged from the back on August 22, 1932. About 150 g. of paraffin were inserted. Pneumolysis was fairly easy in all directions, especially medially, on account of the tissue covering and the laterally situated cavity. The loosening process presented difficulties only towards the front and side, in other words where the cavity lay nearest the surface; all difficulties, however, were overcome. Thus the plombage laid on a bed fixed by the interlobar space. Expectoration before the operation amounted to 30 c.c., and contained bacilli and elastic fibres. Fourteen days after the operation all signs of sputum had vanished. Post-operation progress, with the exception of usual temperature-curve, of no importance.

Exairesis had been performed to reduce the diseased upper lobe, which was not sufficiently demarcated. In a relatively short time, helped by a favourable position, it formed a resistant base for the plombage and caused the area with small cavities to disappear. Thus the plombage, which proved a complete success, was made possible.

Owing to difficult family circumstances this lady could not decide to undergo the fairly lengthy cure which was absolutely necessary. The extremely unsettled mode of life of her family, who, unfortunately, in spite of my warnings, took no consideration for her state of health, quite nullified the good effects of the operation. The stationary condition on the contralateral side became disturbed, and basal foci flared up in the original cavity-area, so that after just over a year, the post-operation condition, which had been good in the first place, naturally grew worse, in spite of the satisfactory local action of the plombage.

CASE 5.—A man of fifty-two. Catarrh with expectoration for years. Never under medical treatment. For the first time, in September, 1930, a right-sided pulmonary-tuberculosis with cavities was diagnosed. A conservative cure improved the general condition without being able to affect the amount of expectoration of 40 c.c. with bacilli and elastic fibres. Sedimentation test of the red blood-corpuscles considerably increased. Attempt at pneumothorax without success. Plombage operation on April 9, 1931, from behind through the fourth rib. Amount of paraffin inserted 200 g. Post-operation progress normal, except for suppuration of a suture. The

expectoration decreased to 5 c.c.; was, however, still positive. Nevertheless considerable improvement in general condition, enabling the patient to resume work on July 1, 1931, and afterwards lead an active life and in a very good condition. Several observations gave always the same result. Expectoration in varying amount, 5 to 10 c.c., with few bacilli. Slightly improved sedimentation test of red blood-corpuscles. Sleep and appetite good. Increase in weight. No tiredness. Symptoms of cavities could still be physically detected from the front towards the parasternum. The cavity was diminished on the film, but had sunk medially and downwards by a good intercostal space. An explanation of this change of position can only be that the already deeply situated cavity sank towards its base on account of the loosening of its lateral zone of suspension during apicolysis. Two years after the operation there was a perforation of the plombage into the cavity. For four months the patient had a severe convulsive cough, and expectorated match-like fragments of wax, varying in size, spiral in shape. As the temperature was high only at the beginning, yielding to a simple rest-cure combined with a raising of the general condition, and as several X-ray examinations failed to show an actual cavity, while the expectoration subsided to slight negative remains, the intended plastic of the apex was not performed. The patient has been at work again for the last six months. The last sputum examination was positive, without elastic fibres.

The patient recovered to a remarkable degree in spite of this serious complication. As a matter of fact the pulmonary tuberculosis was not extensive, and indeed, during a certain period, it became a purely local problem. The further development of the case remains to be seen, and it is not improbable that, owing to the recurrence of positive sputum, a partial rib resection may be undertaken, against which the patient has up to now vigorously protested.

Case 6.—This is a lady of thirty-four years of age, with an old, bi-lateral, stationary, but fairly slight apical tuberculosis. An apical cavity of the right lung grew in the course of a few months from the size of a pea to the full size of a mandarine. Pneumothorax treatment had no effect owing to extensive apical adhesions. In spite of an extensive adhesion of the base of the lung to the diaphragm, the phrenicotomy operation failed. The patient had constant temperatures, and although at first her state of nutrition was fairly good, her condition seemed very doubtful. Sputum 30 c.c. with elastic fibres. Blood tests bad. The usual conservative treatment was quite useless. The cavity grew, and in addition, soft bronchogeneous spots appeared on the other side, so that the outlook became very serious. After careful consideration, the plombage operation was resorted to as a last hope, to try and arrest the alarmingly rapid progress of the disease. The operation was successful, and yet the course of the disease remained uninfluenced. A primary collapse of the cavity itself could not be brought about owing to the extensive tuberculous infiltration of its walls, and it still remained gaping widely, pushed downwards and forwards by the plombage material. Nothing could arrest the progress of the disease, and a few months later the patient died of it.

From the very beginning we were aware of the unsatisfactory indication. But having exhausted all conservative and medicinal modes of treatment, we made an attempt, in spite of the desperate situation, at least to strike at the principal focus of the disease, but unfortunately without success.

The patient suffered from a mental malady as well as the tuberculosis. Her mental state became worse to a remarkable degree before the pulmonary tuberculosis was diagnosed by clinical methods. It is difficult to say to what extent the two diseases reacted on each other. In a general way, however, it must be said that mental troubles, especially those of a depressing nature, are more likely to be occasioned by tuberculosis than the reverse. In the course of years I have been able to prove this *ex juvantibus* many times over. Often in treating tubercular patients who seemed clinically to be in a stationary condition, and who were suffering at the same time from mental trouble, I have put them into the hands of a specialist. Later on these mental disturbances found a simple explanation in a fresh outbreak of the disease, and vanished when the tuberculosis had been duly treated by the correct therapeutical methods. I have never known pulmonary tuberculosis to be cured by psycho-therapy alone, and all the examples which are quoted fail to bear investigation, which is not remarkable when you consider the extremely varied course which tuberculosis may take. This does not mean that mental manifestations should not receive every possible attention, but I do believe that it is more important to use careful judgment and to confine the treatment to the tuberculosis, than to make use of the psycho-therapy which is nowadays so much in favour.

In seeking the reasons for the failure of the operations, we must deal with the cases separately. With the exception of Case 5, in which, by a technical error, the primary cavity-collapse did not succeed at first, resulting in subsequent perforation, there was no actual relapse in any of the other cases, but a subsequent advance of the disease. The indication was clear in Cases 1, 2, 3, and 4. In Case 6 we were aware of the uncertainty of success, owing to the general, progressive lung process. Unwillingly, and with the sole idea of trying to help, we decided on the operation, which the patient bore well, but without deriving any benefit from it. Thus we see that in cases of feverish, progressive cavity-tuberculosis of the lungs, where there is no demarcation of the cavity-area, a plombage operation should not be attempted, even if the topographical conditions appear favourable. An operation is a much too primitive method to be able to influence a general unfavourable process.

In Case 5 an apical plastic operation was considered, but the patient strongly opposed this on the grounds of being an elderly man and father of a family, and therefore not wishing to incur even the slightest risk. There is no doubt that a plombage is much more easily borne than even a partial rib resection. The post-operation progress is also considerably

simpler, so that in this respect the plombage is certainly to be preferred. I know that other writers report contrary experiences, and these, in my opinion, can be explained not so much on purely technical grounds as from irregular indication. Over and over again we are astonished at the good post-operation condition of patients who stand an operation without any signs of shock, which occurs only in a limited number of cases of plastic operations. In Cases 1 to 4 there was no question of a plastic, mainly because the other side was too seriously affected, and Case 1 had a defective heart as well, which in itself was sufficient to render an operation impossible. Moreover, the indication here was a foregone conclusion, because with these four patients the disease was in an interval-stage, and manifested, if only temporarily, a more or less local problem.

In Cases 1, 3, 5, and 6 a primary cavity-collapse was not achieved. In Case 1 the right apicolysis was technically impossible whether medially, from the back, or from above, and could not even be completed by a further operation from the front. It could not be forced owing to the danger of tearing the cavity. But the good results, which lasted for two years, prove that even a partial cavity-collapse may in certain cases be regarded as successful. Either one must let this suffice, or, if the ensuing improvement in general condition warrants it, one follows it up with a plastic, after removal of the plombage material, thus achieving the desired complete closing-up of the cavity. So that a preliminary plombage, although we have first to wait and see if it is successful, can be used to prepare a cavity for a subsequent plastic, which could not be performed in the first instance owing to the uncertain general condition of the patient.

In Cases 3 and 5 there was a technical error, such as is always liable to occur. In contrast to Case 1, where it was a question of "too little," the apicolysis here went too far. Prior to performing the operation, therefore, the position and basal zone of the cavity should be clearly ascertained, in order to avoid unpleasant consequences. Otherwise the cavity will sink medially, because the operating surgeon naturally tends to push forward the pneumolysis in the direction where it goes easiest, namely laterally. In Case 3 the cavity could only be closed after the operation by means of a temporary paralysing of the diaphragm; yet even after this, some old foci at the lower edge of the plombage flared up, detracting to a certain degree from the success of the original operation. In extensive phthisis you have always to reckon with a certain percentage of relapses which cannot be traced back to faulty operation, because this latter only aims at influencing a narrowly defined area. The renewal of activity in old foci under the plombage is just one of those occurrences that can arise sometimes, so that one may wonder whether it would not be better, in cases that are somewhat

uncertain, to extend the plombage beyond the actual cavity-area. The success of a subsequent paralysing of the diaphragm is too uncertain to be relied on.

Case 5 calls for a few additional remarks. The fact of the pneumolysis reaching too far laterally drew the cavity medially downwards, in which position it remained quiescent for two years. The patient had full working capacity with very little positive sputum. The perforation which occurred later started with great violence, gradually subsided, and in the course of four months the paraffin was all coughed up. About two months after the perforation began the patient resumed the regular cure, although for the reasons already stated he opposed the suggestion of a plastic.

When a perforation occurs early, it generally requires prompt action (removal of the plombage, partial rib-resection) owing to phlegmon of the wound-tissue. But in the case of later perforation, if its progress is not too violent one may take up a more or less waiting attitude. This I did with another case of late perforation in which the plombage had been inserted in a different place. The perforation was torpid from the outset. Here, too, the plombage was superimposed on a large, imperfectly collapsed cavity. A plastic was impossible owing to the precarious general condition and the uncertainty with regard to the other lung. At the same time the operation had such a stimulating effect on the organism that a year and a half later it became advisable to remove the plombage and perform a plastic on eight ribs, which brought about a collapse of the cavity, with good lasting results (four years now). Late perforations have generally to be subjected to an apical plastic, which is often accompanied by good results owing to the stabilising effect on the body and the strong local production of connective tissue stimulated by the foreign bodies in the paraffin.

Moreover, Cases 1, 2, and 4 exemplify very strongly the first principles of the subsequent conduct of all patients who have suffered from pulmonary tuberculosis: regular habits, good food, and access, according to need, to strictly supervised treatment. The treatment of pulmonary tuberculosis by operation is only one factor, though admittedly in many cases the most important. But it must not be over-estimated, either by doctor or patient. A regular mode of living—we have proved this again and again—often permits of the full performance of duties. But as soon as it becomes a habit to infringe the rules of health, tuberculosis, the general disease, reappears and undoes the good result of all our care and patience.

In glancing at the tendencies of modern collapse-therapy we often feel inclined to ask whether the natural, unalterable course of things may not sometimes be upset by laying too much stress on the value of technical

intervention. The tuberculosis specialist who is anxious to widen the sphere of his labours must often be forcibly reminded of the divided nature of his methods of treatment. It is for each doctor to find out the individual path for his patients according to his knowledge, his power, and his temperament.

ON PERIFOCAL AND TRAUMATIC HÆMORRHAGES IN PULMONARY TUBERCULOSIS

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According to the classical doctrine expounded by Sir James Kingston Fowler there are three common causes of pulmonary hæmorrhage in tuberculosis:

1. The rupture of an aneurysm of a branch of the pulmonary artery within a cavity.
2. The rupture of a pulmonary vessel the wall of which has become tuberculous and eroded to such an extent that free communication between the blood stream and the air spaces of the lung is established.
3. Small capillary hæmorrhages in all parts of the lungs, identical with the "parenchymatous hæmorrhages" of other authors and which, according to Baldwin, Gardner, and Petroff, are slight and cannot be traced to a single source.

A survey of the literature shows that the cavity aneurysm is the only generally recognised anatomical basis of pulmonary hæmorrhage, which, nevertheless, in the majority of cases cannot be assigned to this source. Other causes suggested are: (1) An erosion of smaller branches of bronchial arteries by recently developed foci (Fishberg, Oeffner); (2) a dilatation and distortion of vessels within fibrotic and shrinking areas of cirrhotic tuberculosis (Ellmann); (3) local congestion and a general oozing (diapedesis) from the engorged vessels (H. Morriston Davies).

To these suggestions one of us has been able to add three anatomical findings which give evidence as to the cause of pulmonary hæmorrhage

in cases of tuberculosis without aneurysm or tuberculous vasculitis (Pagel, 1924, 1926, 1930):

1. Varicosity of bronchioli, bronchiolectases and alveolarectases within fibrotic tuberculous tissue due to chronic local or general blood stasis. The formation of varices appears to be a sequela of the tuberculous process, a "metatuberculous" lesion, which is identical with that developing during a non-tuberculous fibrosis of the lung, such as bronchiestasis, chronic pneumonia, cardiac blood stasis, etc. The formation of varices in the walls of the bronchioli and dilated alveoli explains hæmorrhages in cases of fibrotic tuberculosis of long duration and also those of the so-called initial hæmorrhages, which are due to the organisation and fibrotic conversion of early post-primary foci, especially in the apex of the lungs.

2. Perifocal hæmorrhages occur as circular areas of hæmorrhage around tuberculous foci, either within the surrounding alveoli or between the layers of the capsule. They are due to a toxic alteration of the vessels in hypersensitive tissue, and are among the phenomena of allergy. They can be reproduced in animal experiment—*e.g.*, by injection of old tuberculin in tuberculous, and therefore hypersensitive, guinea-pigs, which results in the so-called tuberculin shock, a fatal paralysis of the vessels accompanied by extreme hyperæmia and hæmorrhage within the organs, particularly in the environment of tuberculous foci (Fig. 5A). Such changes in the vessels within or around the foci, due to exogenous introduction of the specific toxin (the antigen), are referred to a reaction of the antigen with antibodies formed in the foci, a reaction which may also result from an endogenous increase of the antigen in the organs or humours. It is generally recognised that allergic reactions of the tissues to every kind of antigen (horse serum, foreign protein, etc.) exert selective influences on the vessels and their nerves with resultant vascular dilatation, paralysis or spasm, and a consequent hyperæmia, exudation, or necrosis. The highest degree of exudation leads to a permeability of the vessel to all its contents, particularly red blood-corpuscles, and hence to hæmorrhage. In tuberculosis perifocal hæmorrhages, which therefore reveal an allergic hypersensitiveness of the tissues to tuberculous antigens, may attain to a remarkable size and explain phenomena of transitory perifocal infiltrations from which the circular caseous focus develops. In cases of early infiltrations such perifocal hæmorrhages may be the anatomical basis of initial hæmorrhage.

E. Brieger drew attention to the occurrence of perifocal hæmorrhage in certain cases of a special form of pulmonary tuberculosis with acute dyspnœa, due, according to the French authors, Letulle and Besançon, to a rapid outspread of recent tubercles.

In some cases an auxiliary influence of blood stasis has seemed probable

(Simonson). But the importance of the toxic factor was emphasised both by the French authors and by Brieger.

3. Hæmorrhages from liquefying foci. Liquefaction is due to a hydration of a caseous focus by exudation of blood serum and plasma from the vessels. This is usually followed by an exudation of leucocytes and monocytes leading to decomposition and absorption of the focus and its transformation into a cavity. It has been proved by animal experiment that liquefaction also must be regarded as allergic reaction (Roemer, Pagel, E. R. Long) dependent upon vascular alterations analogous to those caused by specific antigen-antibody reactions. Such allergic reaction may be excited by non-specific irritants (sunlight, X-rays) or pathological conditions (diabetes, asthma), which alter metabolism and so convert the tissues into a suitable culture medium for tubercle bacilli. These, after proliferation, act as antigen within an allergic tissue and excite the allergic reaction, according to the intensity of which exudation of serum, fibrin, leucocytes, monocytes and eventually of red blood-corpuscles occurs, and, when the last predominates, hæmorrhage may develop.

Recent liquefaction in tuberculosis may also lead to pulmonary hæmorrhage in a more mechanical way, when liquefaction develops so rapidly that vessels are involved without previous alteration of the walls, and communication between the blood stream and the air spaces is established.

Thus, hæmorrhage in pulmonary tuberculosis has various causes, which may be differentiated according to their relation to the focal tuberculous process. The varices are independent of the latter, being rather a consequence of non-specific fibrosis and cicatrisation. Similarly, the aneurysmal hæmorrhage is accidental, and not directly connected with the tuberculous process, since the aneurysm develops as the result of the gradual corrosion of the external parts of the vascular wall within a cavity. On the other hand, perifocal hæmorrhages, and those occurring during liquefaction, are closely associated with the development and fate of the tuberculous process.

The varying and complicated mechanism of pulmonary hæmorrhages in tuberculosis has hitherto received inadequate attention, and it now seems desirable to collect cases providing anatomical proof of hæmorrhages, in which there is no evidence of the simple rupture of a cavity aneurysm or of a vessel with tuberculous involvement of the wall. The following is one such example:

Patient aged twenty-seven. Father died of tuberculosis, 1928. Onset of disease, 1928, signalised by hæmoptysis and occasional slight rise of temperature. Hæmoptysis repeated one year later. General condition good, in spite of dyspnœa which continued for several years. A series of X-ray pictures shows that the tuberculous process began with the formation

of a thin-walled cavity in the right upper lobe followed by the development of daughter foci in the right upper and left middle fields of the lungs, apparently due to aspiration after the first hæmorrhage. Rapid liquefaction and "early cavity" formation in one of these daughter foci justify, in this case, the assumption of high specific hypersentiveness. Fatal hæmorrhage, April, 1935.

The autopsy revealed two thin-walled cavities, each the size of a small plum, one in the right upper lobe and one in the lower part of the left upper lobe. Each cavity was filled with a blood clot. When the pulmonary artery was opened from the arterial conus downward to the smallest branches, neither aneurysm nor other source of hæmorrhage could be detected. In addition to the cavities mentioned there were, in various parts of the lung, numerous caseous and partially liquefied foci the size of a cherry.

Associated with these foci were peculiar local hæmorrhages, which, in our opinion, constitute the specially interesting feature of the case. Each hæmorrhage took the form of a ring or cushion (Fig. 1), either surrounding the focus or situated between the capsule and the caseous and liquefying mass. Similar perifocal rings of hæmorrhage were observed within the walls of the cavities (Fig. 3), and in some caseous bronchitic foci the cheesy mass was impregnated with blood, so that its colour was pinkish-yellow, darkening at the margin to deep red (Fig. 2).

Histological examination showed:

1. An abundance of capillaries in the marginal areas of the tuberculous foci (Fig. 4), even small epithelioid-cell tubercles exhibiting fine capillary spaces filled with red blood-corpuscles.

2. The perifocal and intrafocal hæmorrhages obviously emanated from the capillaries both of the internal layers of the capsule and of the marginal areas of the caseous foci, for these capillaries were filled with red blood-corpuscles to bursting point (Fig. 4).

3. Blood aspiration as a possible source of these hæmorrhages could be definitely excluded, because it was evident that the blood had been forced into the caseous material from the margin of the focus (Fig. 5) and not from the centre to the periphery, which would have been the case had the hæmorrhage been due to aspiration.

The distribution of the hæmorrhages was excellently demonstrated in detail when acid fuchsin was employed to stain the red blood-corpuscles, according to the methods of Mallory, Laidlaw or J. Abello-Pascual. The latter author has pointed out the influence of the increase in the numbers of capillaries and vessels in tuberculous foci on the transmission of tubercle bacilli to the blood stream and hence to other parts of the lung and to other organs of the body. By means of Laidlaw's method, hyperæmic capillaries were easily shown not to be confined to the margins of the foci; even the

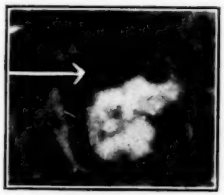

FIG. 1.

Small liquefying caseous focus with large hæmorrhage between caseous centre and fibrotic capsule (arrow).

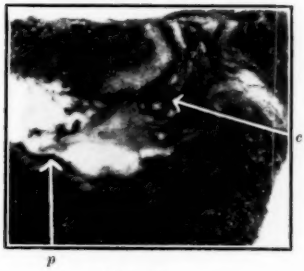

FIG. 2.

Caseous bronchitis. The caseous material mixed with blood (*c*). Perifocal hæmorrhage between wall of the bronchus and pulmonary tissue (*p*).

FIG. 3.

Perifocal hæmorrhage within the wall of a small cavity (arrow). Recently liquefied focus.

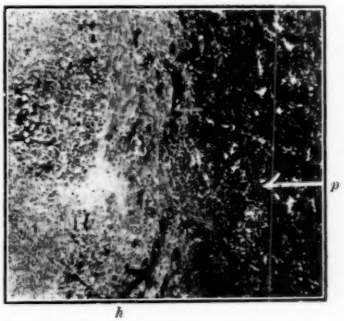

FIG. 4.

Perifocal hæmorrhage. Histological picture: (*p*) perifocal hæmorrhage; (*h*) capillary hyperæmia in the capsule and the caseous focus. Laidlaw staining.

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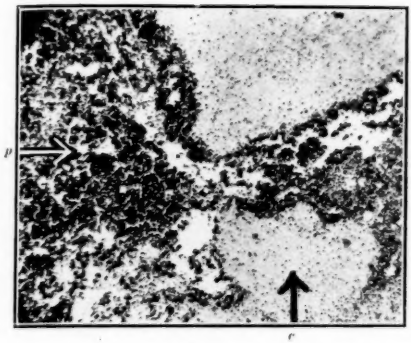

FIG. 5.

Perifocal hæmorrhage (*p*) penetrating into the caseous part of the focus (*c*), splitting it into two.

FIG. 5A.

Spleen of a tuberculous guinea-pig, killed by intraperitoneal injection of 0·2 c.c. of a 1 : 100 solution of Old-Tuberculine. Tuberculin shock. Peri- and intra-focal hæmorrhages (*ph*).

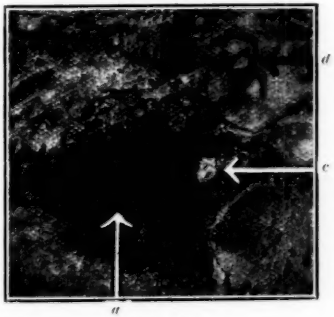

FIG. 6.

Traumatic hæmorrhages with tuberculous foci in the centre: (*c*) calcified nodule with large surrounding hæmorrhagic area (*a*); (*p*) small caseous focus with perifocal traumatic hæmorrhage.

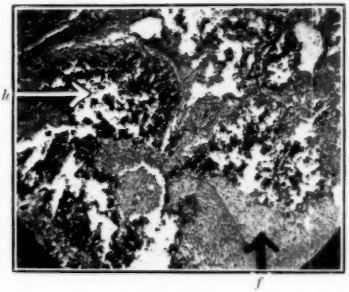

FIG. 7.

Post-traumatic hæmorrhagic destruction (*h*) of a caseous focus (*f*). Laidlaw staining.

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liquefying areas themselves contained capillaries and had islets of hæmorrhage in their environment.

Comment: The perifocal and intrafocal hæmorrhages described are a peculiar phenomenon, which has not been observed in other cases of fatal hæmorrhage, and therefore cannot be ascribed to such conditions as (1) dilatation of the pulmonary vessels in the neighbourhood of a ruptured cavity aneurysm, or (2) dyspnœa caused by the filling of the bronchi with blood after a large cavity hæmorrhage.

Histological examination excluded aspiration as the origin of the hæmorrhages. Although hæmorrhages of similar appearance may occur in caseous foci in the environment of a cavity with aneurysmatic hæmorrhage, these latter may easily be differentiated from those under discussion by the absence of capillary hyperæmia and hæmorrhage at the margins of the foci.

The great importance of capillaries and vascularisation in the development and form of tuberculous foci is emphasised by our case.

The perifocal and intrafocal hæmorrhages, in this instance, were so numerous that they undoubtedly played a part in the development of the fatal hæmoptysis, the immediate cause of which may have been the rupture of a small aneurysm within one of the thin-walled cavities, a condition which could be detected by histological examination.

Both case and history and X-ray pictures indicate a considerable degree of hypersensitiveness, which was expressed by the general tendency of the foci to liquefaction and subsequent hæmorrhage. Moreover, since the cavity containing the aneurysm showed extensive processes of recent liquefaction, we may assume that the aneurysmatic hæmorrhage was similarly a product of recent liquefaction and was closely connected with the hæmorrhages from the other foci. Which particular factor was the precipitating cause of the fatal hæmoptysis cannot at present be decided.

The case does not present similarities to that described by Letulle and Bezançon, wherein the perifocal hæmorrhages due to hypersensitiveness were associated with the rapid dissemination of small foci and hence with acute dyspnœa, and not as, in this instance, with the liquefaction of larger pre-existing foci.

In conclusion: The case of fatal hæmoptysis in pulmonary tuberculosis we have discussed did not show the simple condition of the rupture of a cavity aneurysm but disseminated perifocal and intrafocal hæmorrhages, which must be referred to allergic hypersensitiveness associated with the process of recent liquefaction. A small aneurysm, only microscopically visible in an early cavity, like the capillary alterations in the other foci, appeared to have developed rapidly during recent processes of liquefaction and therefore was not to be regarded as accidental.

II

Among the causes of pulmonary hæmorrhage in tuberculosis the traumatic lesion is seldom mentioned. Although occasionally accidents have been reported as causing fatal hæmorrhage in tuberculosis (Blum, Gegetchkori and Zahradnicky), the anatomical findings in such instances have never been described. A study of the condition of tuberculous foci, after a traumatic lesion, seems to be most desirable, because, in the majority of cases of post-traumatic pulmonary tuberculosis, the possibility of a disturbance of old quiescent foci by the traumatic lesion has to be taken into account. The theory that a traumatic lesion creates a locally lowered resistance to the tubercle bacillus, which may be present in the blood stream and settle at the site of the injury, is gradually being abandoned in favour of the idea that old quiescent or clinically latent tuberculous foci are disturbed and mobilised (Zollinger, von Meyenburg). It is now generally recognised that the majority of persons bear not only the old primary complex but also postprimary hæmatogeneous foci, which, like the former, are usually in a state of quiescence, although they may revive at any time under the influence of debilitating conditions. But hitherto an anatomical basis of the traumatic disturbance of such foci has never been reported.

The following case, although the patient died some hours after the injury, gives opportunity for examination of the effects of a severe trauma on quiescent tuberculous foci, and hence for discussion of the probability of their post-traumatic recrudescence.

Male aged thirty. Hæmoptysis, 1926. Occasionally tubercle bacilli in the sputum until 1929. Since 1933 sputum negative. Worked full time during recent years and was no longer under active treatment. Killed in motor accident, April, 1935. Autopsy revealed extensive subarachnoideal and ventrical hæmorrhages with laceration of the basal ganglia, especially on the right side. No fracture of the skull, ribs, or vertebral column. Large subpleural hæmorrhages. Areas of recent hæmorrhage scattered all over both lungs. Right upper lobe fibrotic. Two larger old caseous foci, each the size of a cherry, in the region of the apical and horizontal branches of the dorsal upper bronchus. Each focus was surrounded by a thick fibrotic capsule and showed a perifocal hæmorrhagic ring. Outspread of smaller calcified nodules all over the left upper lobe. One larger calcified nodule occupied the centre of an area of diffuse hæmorrhage (Fig. 6), in the neighbourhood of which were two smaller old caseous foci with perifocal hæmorrhage (Fig. 6). In the left apex a typical discrete miliary outspread.

Histological examination demonstrated that the hæmorrhages were not

confined to the non-tuberculous lung tissue, and, in the tuberculous regions, to the perifocal areas, but that they had also penetrated into the old caseous and calcifying foci, parts of which were entirely destroyed and replaced by red blood-corpuscles (Fig. 7). Whereas completely calcified foci had resisted traumatic disintegration, the destruction of the old caseous and calcifying foci was much more impressive than that of the lung tissue without tuberculous foci. In the latter, capillaries and alveolar spaces were extremely dilated and filled with red blood-corpuscles, the interalveolar septa not being destroyed to any greater extent. There were also some hæmorrhages confined to perifocal areas, but the majority of the pulmonary hæmorrhages had nothing to do with the tuberculous foci and were scattered all over the organ.

Comments: The results of an injury in this instance were hæmorrhagic destruction of old caseous foci and perifocal hæmorrhages. In contradistinction to the former case described, the majority of the pulmonary hæmorrhages had no relation to the tuberculous process, for, although there was no fracture of the ribs or vertebral column, the traumatic lesion undoubtedly accounts for them all. The experimental and clinical observation of post-traumatic pulmonary hæmorrhage without rib fracture (Kuelbs and Gegetchkori) can therefore be confirmed. The tuberculous tissue, including old caseous and calcifying lesions, appeared to be more fragile and susceptible to hæmorrhagic destruction than the normal pulmonary tissue, the latter showing the highest degree of destruction in the environment of the tuberculous foci, as exemplified by the perifocal situation of some of the hæmorrhages. Although the accident led to the almost immediate death of the patient, the anatomical findings in themselves are evidence of a probable revival of the quiescent foci containing tubercle bacilli as the result of their disturbance by the traumatic lesion, and provide the anatomical basis for those cases where the patient survives and where, consequently, anatomical proof of the effects of the trauma is out of the question.

Thus we have the anatomical proof of the theory of Parkes Weber, according to which, as the result of contusional injury, a latent tuberculous focus may be " squeezed or torn in such a way that tubercle bacilli are mechanically disseminated over the neighbouring lung tissue."

In conclusion: This case permitted an examination of the influence of a trauma on old tuberculous foci, demonstrating the hæmorrhagic destruction of the latter. The tissue of the foci appeared to be more fragile than the normal pulmonary tissue, the latter showing the most marked destruction in the neighbourhood of the foci (perifocal hæmorrhage).

Summary.

Two cases of unusual pulmonary hæmorrhage in tuberculosis have been described. The first has shown general perifocal and intrafocal hæmorrhages, which could be referred to focal liquefaction and therefore to allergic hypersensitiveness; the second has demonstrated the results of an injury as seen in the hæmorrhagic destruction of old caseous and calcifying lesions. Proof of the peculiar fragility of the focal tissue and anatomical evidence of the processes stimulating a post-traumatic revival of quiescent tuberculous foci have also been given.

We wish to acknowledge our indebtedness to Mr. E. W. Groves for kind assistance.

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SUB JOVE FRIGIDO

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A DISCUSSION of the use and misuse of sanatorium treatment will degenerate into a mere quibbling with words unless a very accurate definition is made of the sense in which the words " sanatorium " and " sanatorium treatment " are to be used. The word " sanatorium " may have a variety of meanings,

but it is used throughout this article to describe an institution, situated in the country, so designed that the patients treated there shall be exposed to the open air during the whole twenty-four hours, and incidentally to the clemencies or inclemencies of the weather with much less than the usual protection and the minimum of help from artificial heating. " Sanatorium treatment " indicates residence for a considerable period of time in such an institution, with all the exposure to the open air that it implies, coupled with obedience to a very strict routine which enforces regular amounts of rest and exercise, and almost completely controls the patient's mode of existence throughout the whole twenty-four hours: the essential features of the treatment being a disciplined regularity of behaviour, exposure to the open air, long hours of rest, and graduated exercise. From the earliest days of sanatorium treatment it has been the open air that has been the most strongly emphasised of these essentials, and to be at rest in the open air was known as " curing " or " taking the cure."

The sanatorium, then, is a hard place, lacking in personal comforts and luxuries, and if it is designed on orthodox lines the provision of these for special cases is difficult. Since a large proportion of the patients are up all day, and most of them feeling extremely well, they have to be amused and occupied, and it follows that, without being rowdy, the sanatorium is a distinctly noisy institution in comparison with a hospital, where the sick are treated in peaceful quiet. The clumping of boots, the chatter of bodies of patients going about their appointed tasks, and the sounds of the necessary gramophone, wireless, and laughter of the recreation hours are an inevitable part of its routine life. Being situated in the country it does not carry the personal or material equipment considered necessary in a modern hospital, and is also, for the same reason, not easy of access to friends or families who wish to visit the patients.

This method of treatment of pulmonary tuberculosis had an ætiological basis, and its introduction was a response to the recognition of the fact that, apart from infection, the disease was caused by the unnatural habits forced on man by the conventions of civilisation, and that a return to more natural conditions of life under medical supervision would allow the body's natural resources to arrest its activity in a certain proportion of cases. The introduction of sanatorium treatment was a great advance, giving excellent and lasting results in a certain proportion of cases, supplying a definite element of hope in a disease in which, up to then, a fatal termination was only a matter of time. Apart from the good results it produced in the individual, by its grouping together of cases of pulmonary tuberculosis under expert medical supervision, it encouraged and permitted an intensive study of the disease over long periods in particular cases. From the opportunities thus

afforded have come the advances in the knowledge of tuberculosis and its treatment that have taken place in the last forty years.

Exactly how sanatorium treatment benefited tuberculosis remained obscure, but the good results were there and several hypotheses were put forward to explain them. Different physicians modified the detailed technique of the treatment to suit their own ideas, but no universal agreement or satisfactory explanation was reached, and sanatorium treatment became invested with some magic power over this particular disease. The profession and the public were dazzled by its successes, which, as has been pointed out, were something new, and apparently agreeing to ignore both the failures and the temporariness of what appeared to be good results united in a belief that was formulated in a sort of slogan: " This patient has consumption. Send him to a sanatorium. That is the cure for consumption."

Up to 1910 sanatorium treatment was only for the rich and for a lucky few of the working classes, and so it was in the spirit of that universal belief that those who framed the National Health Insurance Act of 1911 decided that this panacea for consumption should be provided for all. This decision was another important step forward, for, perfect or imperfect as it might be and expensive though it was, up to that time sanatorium treatment was the only treatment that had given any good results. But the actual wording of the Act, whereby " sanatorium benefit," which did not necessarily include treatment in a sanatorium, was to be provided when necessary for every sured person, tended to strengthen the popular belief in the mysterious efficacy of sanatorium treatment.

In the last twenty years our knowledge of the ætiology, pathology, and treatment of tuberculosis have advanced far, and it is time that this mystery which has surrounded tuberculosis and made it a disease apart, and the supposed magic power of the sanatorium, were reviewed in the light of this advance.

I would suggest in the first place that sanatorium treatment is not a treatment at all, in the true sense of the word, at any rate of active tuberculosis. The sanatorium course is still a necessity for a large proportion of cases during certain phases of their disease, but its proper place, use, and limitations in the treatment of this disease should be defined and admitted. In attempting this task I think it will be helpful to make an arbitrary clinical classification of the disease into three phases, thus:

Phase I. Active phase. The period during which the lesions are spreading, or if not actually spreading are in such a condition as to produce ***progressively*** pronounced symptoms and ***progressive*** physical deterioration.

Phase III. Quiescent phase. The period during which the lesions are static or even shrinking, and during which, if there are symptoms, these are not pronounced or *progressive*, and during which there is no *progressive* physical deterioration.

Phase II. The period that intervenes between Phase I. and Phase III.

The treatment of Phase I. should be an active and reasoned attack on the disease, whose degree of activity and extent have been accurately assessed. We have surely got past the old " hit or miss " method of putting the patient into a sanatorium and hoping for the best. This phase demands the same treatment as the active stage of any other infection, and, being a grave and dangerous infection, we may say it needs, for want of a better descriptive phrase, hospital treatment. It demands quiet and complete and continuous rest, full nursing facilities, warmth and protection, easy access to all therapeutic and diagnostic auxiliaries, such as departments for radiology, pathology, laryngology, surgery, etc. These are the essentials of the treatment of the active phase whatever additional methods of attack may be used, and surely they can only be supplied in a fully equipped modern hospital. A sanatorium built, equipped, and run on accepted sanatorium lines is about the last place in which such treatment should be attempted.

Those patients in Phase I. whose disease fails to respond to such treatment are obviously unsuitable for sanatorium treatment. But what of those whose disease does respond? Is the sanatorium course always a necessity for them? Apart from the obvious unsuitability of a sanatorium for the case in Phase I., I think it may be well worth while to discuss its necessity and suitability for Phase II. and Phase III. In Phase II. its necessity will depend upon certain factors:

1. Character and temperament.
2. Social and financial environment.
3. Efficiency of home treatment.
4. Type of disease and treatment already given.
5. Need for education.

Before considering these factors in detail it may be agreed that a three months' routine sanatorium course will benefit the physical condition of any normal adolescent or adult up to the age of forty-five, therefore there is no doubt that the average well-chosen case of tuberculosis should show marked improvement under such a routine. But this obvious surface improvement may have but little relation to the actual state of the tubercular lesions, and there is no need to be hoodwinked by this into thinking that it implies that the sanatorium course is a specific treatment for this one disease.

Character and Temperament.—Without mechanical interference the retrogression and disappearance of activity of a spreading lesion are slow and, when obtained, the quality of the quiescence is difficult to gauge. The symptoms of pulmonary tuberculosis are apt to improve or disappear more rapidly than the activity of the lesion, and to fail to reappear much more slowly than does reactivity. The normal individual will not behave as a sick man unless he feels sick. But prolonged and strictly enforced periods of rest and careful graduation of exercise are necessary in this disease long after the disappearance of symptoms. Experience and the X-ray have taught us this. The humble and sensible patient will trust his doctor, and may voluntarily carry out his instructions at home. The doubting and the foolish will not. Therefore when Phase I. has passed, much of the necessity for the sanatorium during Phase II. depends upon the character and temperament of the patient. The regaining of stability will be much safer, usually more rapid, and always more certain under sanatorium control. But the more sensible the patient the less the actual necessity for such a course.

Social and Financial Home Environment.—This factor is a very important one. Since the regaining of sure stability, where there has been no mechanical interference, is slow, the graduation of rest and exercise must be cautious, closely supervised and very strictly carried out. If this period of treatment is not undertaken in a sanatorium it must be possible for the whole routine of the patient's home to be remodelled: in fact, its routine and that of all its inmates must be disorganised and reorganised to suit the needs of the patient. Further, arrangements must be made for frequent medical supervision. Such demands require not only money, but also great self-sacrifice from the rest of the family. This may be possible for the leisured or professional-class patient, but is quite impossible for the working-class patient, even were his home in a salubrious district and not, as it so often is, in a slum.

Efficiency of Home Treatment.—There is no magic in sanatorium treatment: in certain circumstances it can be carried out in the patient's home. But supposing that the difficulties mentioned in the preceding paragraphs can be overcome there is still the question of the efficiency of the home medical supervision to be considered. The guidance of a patient through this phase may not be difficult, but it does require experience—experience which the average general practitioner can but rarely obtain. It also requires certain clinical facilities, in particular easy access to competent radiology. The working-class patient can presumably count on the skilful guidance of his local tuberculosis officer, but the latter is usually denied the vital help of radiology. The leisured or professional-class patient, unless he can

afford the close attention of a specialist, is running a very definite risk unless this phase is spent in a sanatorium.

Type of Disease and Treatment Already Given.—During the active phase there are two types of lesion that interest us in this discussion of the use of a sanatorium: (*a*) the spreading lesion; (*b*) the lesion that is not spreading but is in such a condition, perhaps because of cavitation or secondary infection, that it is producing dangerous progressive symptoms and incapacity.

To take the spreading lesion first. If this has been treated and controlled by rest and drugs alone without mechanical interference, then the sanatorium period is necessary. But if the spread and the symptoms have been checked by mechanical collapse, they are checked, and where the contralateral lung is free from disease there is no absolute necessity for any other form of actual treatment than convalescence. All the real treatment in such a case should and can be given in a hospital. Where the contralateral lung is involved the case is different. Each lung demands its own treatment, and if there is doubt as to the stability of the second lung then the sanatorium course is necessary.

The lesion that is not spreading presents quite a different problem. It will presumably be a lesion that is dense and well entrenched, and it should be possible, while the patient is in hospital, to make certain that it is not spreading. It may be treated by rest and nursing alone, by drugs, or by mechanical collapse. But what good can the sanatorium course do in such a case? The patient may perhaps improve somewhat in general health as a result, but so also would the doctor who sent him. The sanatorium course will not remove the lesion nor materially alter it. The patient will leave the sanatorium with the same potentialities for ill-health with which he entered it and probably with the same symptoms. If the lesion cannot be successfully treated in the hospital, all that the sanatorium can do for it is to gloss it over and obscure its underlying danger by producing a temporary and completely spurious general physical improvement. If such a lesion has been successfully treated in hospital, the patient should be able to go straight back to his home environment perhaps after a short convalescence.

There is another point to be considered in relation to the suitability of this type of case for sanatorium treatment. As suggested above, these lesions are usually fairly extensive, dense, well entrenched and of some standing. Their possessors are often at, or past, middle age, and in addition to the thoracic rigidity and emphysema so often seen in normal individuals at this age there is usually an added emphysema to compensate for their loss of lung tissue and also, in many cases, an ineradicable focus of secondary infection always ready to attack the non-tuberculous parts of their lungs. For such cases, except during the five summer months, the open air regime,

which cannot be avoided in a sanatorium built as such, is extremely dangerous. Exposure to inclement weather can provoke bronchitis in the emphysematous, and all the more readily if a focus of active secondary infection is present. All superintendents of sanatoria can point to fatal accidents of this sort, and in some institutions the October clearance of such cases is a routine observance.

The Need for Education.—There is this to be said in favour of the "sanatorium for every case" school—that the educative value of sanatorium life is great. It gives the patient a model routine on which to order the behaviour of his home-life, it points out to him the dangers that may beset him and should demonstrate to him the limitations that his particular type of disease imposes upon him, at the same time giving him confidence to live right up to these limitations. Many a patient, after hospital treatment alone, is so impressed by the terrifying possibilities of tuberculosis that he remains virtually incapacitated. He would like to test himself out, but rightly fears to do so unaided. The sanatorium can help him to take this post-graduate course in comparative safety.

These considerations lead to the conclusion that the sanatorium course is no longer a treatment for active tuberculosis and is no longer, as it once was, the only treatment for the disease. It is really a period of protection, testing, and education during that phase of the disease that intervenes between the phase of activity and the phase of quiescence. It cannot remove or alter lesions and, beyond that it teaches the sensible patient how he should live and what his limitations are, it gives no specific, or even any, security against reactivity or further spread of the disease.

The technique of the institutional treatment of pulmonary tuberculosis has improved rapidly during the last fifteen years, and has far outstripped in efficiency the home-treatment of the leisured classes and the dispensary treatment of the working classes. The time has now come, not for the multiplication of sanatorium beds, but for the provision of more hospital beds and, what is more important, the improvement and bringing up to date of home and dispensary treatment. It seems almost unbelievable, for instance, that it should be a rarity for a tuberculosis officer to have free access to radiology. Those responsible for this amazing situation seem to have no hesitation in providing sanatorium treatment for every case at a cost of £120-150 per head per year, but cannot be persuaded to encourage early diagnosis, to help in the selection of suitable cases for institutional or home treatment, or to give the tuberculosis officer the chance of maintaining the good results afforded by such treatment, by providing him with an X-ray plant at the initial cost of the institutional treatment of but four patients for one year and at a maintenance cost equal to that of the institutional treatment

of two patients per year. Home and dispensary treatment have been neglected because of the undue reputation for efficacy which has been credited to the magic sanatorium treatment, a reputation which is unmerited and cannot be supported by facts. If the non-institutional treatment of tuberculosis could be modernised and brought into line with the present-day institutional treatment, the necessity for the sanatorium course, expensive to both State and patient, and distressing to the latter, would be decreased, while its efficiency would be increased by proper case-selection and proper after-care.

BLOOD SEDIMENTATION TEST

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Many of the patients admitted to this hospital have had their blood sedimentation rate tested shortly before admission in some other institution: it has been found that in many cases a similar test on admission here has given a figure in no way comparable to that obtained before admission. This was obviously due to a difference in technique, and one of the most obvious differences was the length of tube used. The effect on the blood sedimentation rate of using varying lengths of tubes is therefore being investigated.

Westergren tubes have been used, the height of the columns of blood being 50 mm., 100 mm., 150 mm., 200 mm., 250 mm. The blood used was venous blood from the ante-cubital veins, diluted 1 : 4 with sodium citrate 3·8 per cent. The length of the column of clear serum was read at hourly intervals.

It was found that the fall of the red blood cells was not the same in each tube in the same length of time. In nearly all cases the fall was greatest in the longest columns: but where the sedimentation rate was small the fall in a column of 250 mm. at the end of one hour was occasionally actually less than in a 50 mm. column. When, however, the red blood cells were allowed to fall for several hours it was apparent that the fall was in proportion to the length of the tube, provided that a similarly proportionate length of time had been allowed. Thus in some cases the reading in a 250 c.c. column at five hours was five times that in a 50 mm. column in one hour. As a rule, however, this was not the case, as the fall in the first hour in all tubes was very irregular, and it was only at the

end of the second hour that the reading in the 50 mm. tube was sufficiently accurate to bear a constant relation to the other columns: then the reading in columns of 100 mm., 150 mm., 200 mm., and 250 mm., were 2, 3, 4 and 5 times that in the 50 mm. column at the end of 4, 6, 8 and 10 hours respectively.

In certain cases, however, no ratio could be found: the reason for this is obscure and is being investigated.

Below is the result of one case chosen as being typical of the great majority of the results.

Length of Tube in mm.	*Hours.*									
	1	2	3	4	5	6	7	8	9	10
50	30	31	33	35	35	35	35	35	35	35
100	43	56	58	59	61	62	62	62	62	62
150	52	81	88	90	92	92	93	94	94	94
200	61	99	115	119	122	123	124	125	125	125
250	61	116	141	150	154	156	157	158	159	159

Column of clear serum at end of each hour in each tube.

For each length of tube there appears to be a length of time after which there is only a trivial drop in the column of red blood cells—for 50 mm. about two hours, 100 mm. four hours, etc.

On p. 41 is a graph of the same case as shown above, which shows well the marked fall in the first few hours, and then the trivial fall thereafter. It is suggested that the marked fall is due to the factors which are measured by the sedimentation rate, and the small fall thereafter represents an effect due to the "packing" of the red blood cells from their own weight.

Thus it would seem:

(i) That the fall is not equal in tubes of different length in the first few hours.

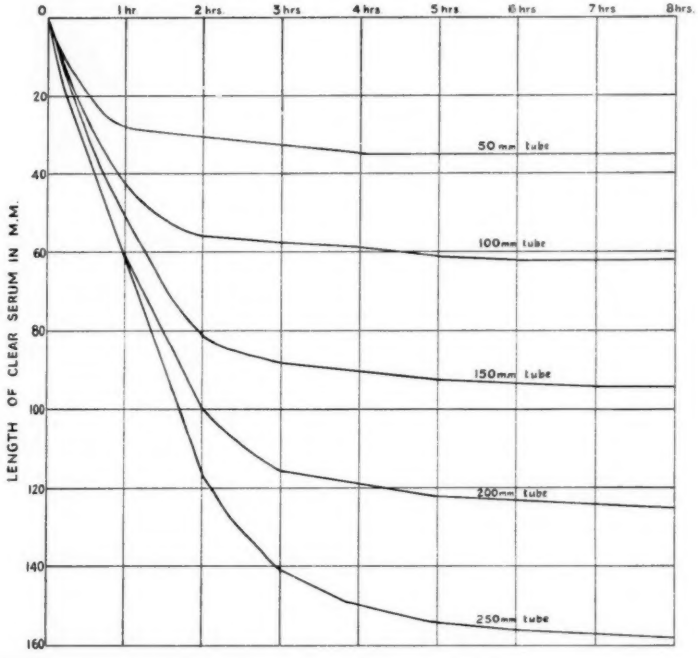
(ii) That the fall is only proportional to the length of the tube when a proportionately long time has been allowed.

(iii) That there is an optimum time at which each tube should be read.

(iv) That an increase of the reading occurs, due not to the factors affecting the sedimentation rate, if the tubes are left longer.

It is thus obvious that a blood sedimentation rate cannot be expressed as a "percentage" at an arbitrarily chosen length of time, but could be so

expressed if the " optimum " time were given for the particular length of column used. Also, since the fall is not regular at first, especially in the longer tubes, a short time is the most suitable as a standard. At present



the blood sedimentation rates from different sanatoria are in no way comparable, and some standard, not too involved, is urgently needed.

I am indebted to Dr. G. Oliver Hempson, the Medical Superintendent of the hospital, for affording facilities for these investigations.

CONSULTATION

CASE

By E. RIST,
M.D.
Physician-in-Chief to the Läennec Hospital, Paris.

Captain F. T., French Army, thirty-nine years old, was sent to me for a consultation by Dr. W. J., of Pau, with the request that I should give my opinion as to the advisability of a thoracoplastic operation, on account of a tuberculous cavity in the apex of the right lung, an attempt to induce an artificial pneumothorax having failed.

He came to see me on October 24, 1935, with the following history. The onset of his disease dates back as far as 1924. He then had a "fistula in ano," which was treated without any reference being made to the possibility of lung disease. And yet, soon after this symptom had made its appearance, he began to cough; he also complained of distress in his chest. For many years he was considered as a neurotic patient, and accordingly submitted to various hydrotherapeutic procedures, which succeeded only in making his subjective feeling of fatigue and dyspnœa worse.

In February, 1931, he fell ill with fever and blood-stained sputum. This episode was the first occasion for a physical and radiological examination of his chest. The upper lobe of both lungs was found to be diseased, with cavities on both sides. Tubercle bacilli were discovered in the sputum. He underwent a course of tuberculin, spent some time in a sanatorium in Montana (Switzerland), and suffered there from a right-sided pleural effusion. His voice became altered and a tuberculous infiltration of the arytenoids was observed. He lived the two following years in Briançon in the French Alps and in Grasse, under rather intermittent medical control; finally he was admitted, on February 18, 1935, to Dr. J.'s sanatorium in Pau.

A radiograph taken at this period and compared with the previous one made four years before showed that the character of the lesions had been considerably modified. The right upper lobe had undergone a remarkable degree of shrinking; its volume was very appreciably reduced; the fissure

had been displaced upwards and its shape was that of an arch, the concavity of which faced downwards. The diseased lobe was very dark; no cavity could be outlined in the dense shadow; but, on auscultation, numerous moist consonant râles were audible. The obliquity of the ribs was exaggerated on the right side, the trachea was displaced to the right. The movements of the right half of the diaphragm were very limited, and the costo-diaphragmatic sinus was obliterated.

On the left side of the chest the fissure was thickened, and above its axillary end the outline of what seemed to be two or three coalescing cavities was visible. But no abnormal sound could be heard on that side except that the vesicular murmur was diminished in intensity.

Captain T. willingly submitted himself to a severe and prolonged rest-cure, with the result that his subfebrile temperature was definitely restored to normal. He gained 11 pounds. The annular shadow in the left lung gradually disappeared. The not very abundant sputum was never free from tubercle bacilli. Sometimes the bacilli were present in small numbers and could only be discovered by concentration methods, but a little before the patient came to consult me in Paris, they had again become abundant—about 30 units in the field of the microscope on direct examination.

Gold treatment had been well tolerated but ineffective. Artificial pneumothorax on the right side had been entirely unsuccessful on account of the antecedent pleurisy, as was to be expected. The patient began to be restless and disappointed. He is an intelligent man. He realises very well that prolonged rest has been of great benefit. His left lung is practically healed. He has no more fever. He gains weight. But the process of spontaneous healing has carried him up to a certain point, and not further. The cavity in his right apex has not been influenced, and remains a permanent and dangerous menace. He is still short of breath on exertion. He is unable to work. Could not something be done in order to bring about a complete, definite cure? Why not try a thoracoplasty?

It was the patient's own suggestion. Dr. J. did not think that it was *a priori* unreasonable. The disappearance of the cavity in the left lung was indeed rather recent. It was true also that Captain T.'s nervousness was an unfavourable circumstance: he was subject to attacks of tachycardia with a feeling of precordial distress, he had strong vaso-motor reactions, sudden pallor followed by sudden flushing, he had a tendency to be impatient and irritable. But this was perhaps not absolutely incompatible with a very cautious succession of small, careful operations. Dr. J. desired my opinion, and sent the patient to Paris.

Captain T. gave me at first sight the impression of being a well-

nourished and even robust man. He was not cyanotic. His pulse and temperature were normal. Dr. J., in his letter to me, had mentioned that the patient was dyspnœic. But I noticed that his dyspnœa was of a particular kind. It was inspiratory, with a slight amount of stridor; this feature was, the patient explained, quite recent, following what had seemed to be an ordinary cold. It was very suggestive of a beginning stenosis consequent to his old-standing fibrous tuberculosis of the larynx.

I noticed on the fluoroscope the dense retraction of the *right* upper lobe, the displacement of the trachea to the right, the small amplitude of the movements of the diaphragm. The middle and inferior lobes were transparent, and gave a normal vesicular breathing. But on the right apex, which was quite flat on percussion, tubular breathing with numerous coarse, moist, consonant râles could be heard; the presence of a cavity, although not visible on the fluoroscope or on the radiographs, was therefore evident.

On the last radiograph, made a few days before, there was no trace of a cavity in the *left* lung, nor could I see any on the fluoroscope. But on a radiograph dating back to September, 1934, two small cavities were still visible above the fissure. In their place I now noticed some very slight nodular shadows. The vesicular murmur was rather weak, but no adventitious sounds were heard.

If one took into account only the condition of the chest, there was certainly something to be said in favour of a thoracoplastic operation. It seemed wise, however, to wait a little longer in order to ascertain more definitely the nature of the scars in the left lung. The volume of the shrunken upper right lobe was reduced to such a degree that the section of the upper four or five ribs would probably suffice to produce a satisfactory collapse. A phrenic evulsion seemed to be out of question.

Nevertheless, I finally pronounced myself averse to the idea of a surgical operation, for the following reasons:

In the first place, the condition of the larynx seemed to me most ominous, the inspiratory dyspnœa being liable to prove a very seriously complicating factor during and after the operation, especially in a nervous and " highly strung " patient.

Furthermore, my attention was drawn by an important symptom connected with the intestinal tract. Captain T. did not complain spontaneously of any digestive disturbance; but, on close questioning, I discovered that for the last two years he has frequently a rather urgent, though painless, desire to empty his bowel, followed by a soft, unformed stool. I found the ileo-cœcal region tender on pressure. The presence of a tubercular ulcerative colitis at its initial phase was highly probable, and, in my opinion, it distinctly contra-indicated any surgical intervention on the chest.

I had, therefore, the painful duty to dissuade the patient from undergoing an operation which he sincerely believed would improve his condition and even restore him to normal health, whereas I was convinced that it would only precipitate an unavoidable but, under cautious management, by no means immediate end.

CLINICAL CASES

ABSENCE OF MOVEMENT ON ONE SIDE OF THE CHEST

By L. S. T. BURRELL, M.D.(CAMB.), F.R.C.P.(LOND.).

A WOMAN of twenty-two was admitted to hospital with a history of cough for six months and of sudden loss of voice and hæmoptysis two days previously. She stated that she had coughed up over a pint of blood, but this could not be confirmed. She had been in several hospitals and had had sanatorium treatment, but no definite evidence of tuberculosis had ever been discovered.

On examination the right side of the chest moved very little with respiration, and she appeared to be breathing almost entirely with the left side; this was less marked, but definitely present during sleep. There was anæsthesia to pin prick over the left side of the body. The voice was hoarse but her general condition was good, and she was cheerful and appeared to be well.

On examination breath sounds were slightly weaker on the right than on the left side, but no other physical signs were found in the chest apart from the lack of movement on the right side. X-ray showed no abnormality, and after lipiodol injection there was no dilatation or blocking of the bronchi shown by X-ray. Bronchoscopy revealed no abnormality, and it was noticed that whilst the bronchoscope was being introduced, although the patient was nervous and inclined to struggle, breathing was almost entirely confined to the left side of the chest. The vocal cords adducted well and nothing abnormal was found in the larynx.

I think this a case of hysteria, and that the story of hæmoptysis is untrue. It is, however, difficult to understand the unilateral respiration. Not one of my students was able to breathe in that way.

POST-OPERATIVE GANGRENE OF THE SKIN

By L. S. T. BURRELL, M.D.(CAMB.), F.R.C.P.(LOND.)

Physician to the Royal Free Hospital and to the Brompton Hospital for Consumption and Diseases of the Chest.

The patient was admitted to hospital with pneumonia following influenza, which had started three days previously. On admission he was toxic and extremely ill, and had signs of pleural effusion on the left side. On explora-

Post-operative Gangrene of the Skin.

tion the liquid was found to be slightly cloudy but thin, and so it was aspirated. Later, it became definitely purulent and an operation for rib resection and drainage was performed. There was some improvement in the patient's general condition at first, but he continued to expectorate offensive sputum, and the area around the wound became infected. This spread in spite of all treatment, until it reached the state shown in the illustration. The edges of the wound were incised and eventually healing began to take place, but the patient died with multiple abscess in both lungs.

MEETINGS OF SOCIETIES

THE TUBERCULOSIS ASSOCIATION

PROVISIONAL PROGRAMME FOR 1935–6

LONDON MEETINGS

November 22nd, 1935.	Tuberculosis of the Upper Respiratory Tract.	Sir St. Clair Thompson.
	Tuberculosis of the Eye.	Mr. H. R. Bickerton.
	Industrialism and Tuberculosis.	Dr. E. L. Middleton. Dr. P. Heffernan.
January 17th, 1936.	After-Care of the Tuberculous in London.	Dr. Noel Bardswell. Dr. J. B. Johnstone. Miss Marx.
	Intestinal Tuberculosis.	Dr. James Maxwell.
February 21st, 1936.	Primary Tuberculosis in Children and its Relation to Meningitis.	Dr. H. H. Scott. Dr. C. H. C. Toussaint.
	The Treatment of Tuberculous Lesions of Bones and Joints.	Sir Henry Gauvain. Mr. G. R. Girdlestone.
May 15th, 1936.	Climate and its Relationship to Tuberculosis.	Sir Leonard Rogers. Dr. Andrew Morland.
	Recent Advances in Thoracoscopy and Adhesion Section.	Mr. J. E. H. Roberts. Dr. F. G. Chandler.

PROVINCIAL MEETING AT CAMBRIDGE

April 2nd, 1936.	Dispensary Organization.	Dr. Hagius van den Berg (Amsterdam). Dr. R. H. Hazemann (Paris). Dr. N. Tattersall (Leeds).
	Cinematography of the Chest.	Dr. Russell Reynolds.

ANNUAL DINNER AT ST. JOHN'S COLLEGE

April 3rd, 1936.	Chronic Miliary Tuberculosis.	Professor Sayé (Barcelona).
	The Capacity for Work in Pulmonary Tuberculosis.	Dr. Maurice Davidson. Dr. L. B. Stott.
	Visit to Papworth Settlement.	
April 4th, 1936.	Cases for Discussion.	Dr. Vere Pearson. Dr. G. T. Hebert.

ANNUAL MEETING.

A meeting of the Tuberculosis Association was held on Friday, November 22, 1935, with Dr. L. S. T. Burrell, the President, in the Chair.

Sir St. Clair Thomson gave an address on Tuberculosis of the Upper Respiratory Tract. He said that whereas some fifty years ago tuberculosis of the larynx was considered such a serious complication that it was even doubted if recovery ever occurred, now 25 per cent. made a complete recovery. He showed a series of slides illustrating cases of advanced laryngeal tuberculosis which eventually got well. Rest of the larynx by keeping the patient on silence was, in his opinion, the best treatment. Failing absolute silence, making the patient speak only in whispers was next best. If, in spite of silence, the disease progressed or failed to improve, treatment by means of the galvano-cautery might effect a cure. Treatment by lactic acid was not satisfactory, and if used without great care might make matters worse. The same applied to treatment by artificial light. Tuberculin had been given a good trial without success, and so far he had seen no benefit from gold. In his experience, he had never seen a case of laryngeal tuberculosis or cancer caused by alcohol or tobacco, but when once a case of tuberculosis of the larynx developed the chances of a cure were 30 per cent. better if the patient gave up smoking. Rest of the lung by artificial pneumothorax often led to a rapid improvement in the larynx.

It might happen that the larynx healed in spite of spread in the lungs and persistence of tubercle bacilli in the sputum, but if the laryngeal condition got worse there was also a spread in the lungs. A spreading laryngeal lesion, therefore, indicated a bad prognosis.

The lupoid type of laryngeal tuberculosis had the best prognosis.

Diagnosis in the early stages may be difficult, but a simple laryngitis was symmetrical and affected the whole larynx; an unsymmetrical or local lesion, especially if in the posterior part, was suggestive of tuberculosis, and in the anterior part of cancer.

Mr. H. R. Bickerton read a paper on Tuberculosis of the Eye. It is, he said, a rare condition, and is chiefly seen as an irido-cyclitis, which could be recognised by grey nodules. Tuberculosis occasionally occurred in the conjunctiva or even the cornea, but was like lupus, and did not tend to be acute. The old-fashioned habit of licking the eyes of children, when the lids were stuck together in the morning as a result of a simple conjunctivitis, or of licking a foreign body out of the eye, entailed the risk of producing tuberculosis. Phlyctenular conjunctivitis might be regarded as an allergic phenomenon allied to Calmette's reaction. He had known it to occur in one case after every injection of tuberculin. Tuberculosis was very rare inside the eye but might occur as a result of injury, and a tuberculous retinitis might be present as part of a general miliary tuberculosis. Irido-

cyclitis or choroiditis tended to improve by simple treatment, and operative measures were usually harmful.

At the evening session papers were read by Dr. E. L. Middleton and Dr. P. Heffernan on Industrialism and Tuberculosis. Dr. Middleton showed a number of slides illustrating the different stages of work in the production of earthenware, china, and cutlery, and emphasised the point that it is the hard stone used in the works that produced silicosis. For all practical purposes silica alone might be regarded as harmful.

Dr. Heffernan agreed that there is only one dangerous dust—silica. He said that dust-retention might produce fibrosis or some blocking of the lymphatics, but silicosis was a separate and distinct disease. In all cases of pneumokoniosis the seriousness of the condition depended on the proportion of silica in the dust. He showed some radiographs, and pointed out the nodular appearance characteristic of silicosis and the absence of striction which may be seen so commonly in chronic bronchial conditions.

In the discussion which followed, Professor Lyle Cummins said that it was work with hardstone having a high silica content which was dangerous. A patient with silicosis might maintain a fair standard of health, but suddenly would begin to lose weight and fail rapidly, and this usually indicated the onset of a superimposed tuberculosis.

JOINT TUBERCULOSIS COUNCIL

A MEETING of the Joint Council was held on November 23, with Dr. Lissant Cox in the chair, and twenty-three members present.

The chief business was the consideration of a report presented by Dr. McDougall on behalf of the Employment Committee dealing with Care and After-care Schemes in Tuberculosis, a truly encyclopædic memorandum concerning every side of care and after-care work in this country and abroad. Various suggestions were made for Dr. McDougall's consideration, and the circulation of the memorandum was left to the British Legion Press.

On behalf of the Institutions Committee Dr. Blackmore presented a note dealing with Dr. Heaf's memorandum on Tuberculosis Institutions and Chest Hospitals. It was resolved that : " This Council is of the opinion that the benefits of major surgery for both tuberculous and non-tuberculous conditions should be available to every patient in need of such treatment, and considers that steps should be taken by local authorities to provide facilities at Tuberculosis Institutions if adequate arrangements have not or cannot be made at other institutions."

On behalf of the Milk Committee Dr. Hawthorne reported that the

National Association for the Prevention of Tuberculosis Conference at Southport had expressed high opinion of the value of pasteurisation in the production of safe milk, and the Ministry of Health Report took the view that this process does not impair the nutritive value of the milk. Dr. Hawthorne commended to the attention of Council the articles of the Agricultural Correspondent of *The Times*.

A pamphlet on Blood Sedimentation Tests produced by the Association of Clinical Pathologists had been circulated to members and was discussed. A Committee comprising Dr. Heaf (convener), Drs. R. R. Trail and W. H. Dickinson, and Professor S. Lyle Cummins was appointed "to consider and report to the next meeting of Council on the standardisation of blood sedimentation tests."

Dr. Brand gave an account of post-graduate courses held this year. One course had been at the City of London Hospital for Chest Diseases, at which there were twenty-two participants. A study tour in Denmark from June 1 to 15 was followed by ten members. Dr. Peter Edwards had held six short intensive courses, and from November 4 to 9 a course had been held at Brompton Hospital. Dr. Brand also sketched the programme he was planning for 1936.

A letter had been received from the College of Nursing asking for advice as to the precautions that should be taken to prevent tuberculous infection among nurses. A Committee was appointed—Dr. Esther Carling (convener), with Dr. Jane Walker and Dr. Watt—"to report to the next meeting on the danger to nurses of tuberculous infection and the precautions that should be advised."

It was decided to invite the Association of Thoracic Surgeons to appoint a representative on the Council.

REVIEWS OF NEW BOOKS

Gynæcological and Obstetrical Tuberculosis. By EDWIN M. JAMESON, B.S., M.D., Attending Surgeon, Saranac Lake General Hospital. Pp. 256. Price 16s. Baillière, Tindall and Cox, London, 1935.

The first part of this work deals with the effect of pulmonary tuberculosis on the physiology of the female genital organs. Premenstrual fever is very common in tuberculous women and of no significance, but pyrexia during the period is often found in those who are not doing well.

In the second part there is a full account of tuberculosis of the genital organs and peritoneum.

Part III. is devoted to a discussion of the problem of pregnancy in tuberculous women. The indications for interrupting pregnancy are considered in detail, and so are the best methods of managing the patient if pregnancy is allowed to run to term.

The problem of lactation and the effect of maternal tuberculosis on the infant are also described.

The book is full of information not only for the gynæcologist, but for all who are interested in tuberculosis, and it can be warmly recommended. It is well produced and clearly printed on good paper, and concludes with a useful bibliography and index.

Chirurgie de la Tuberculose Pulmonaire. By A. BERNON, Lauréat de l'Académie de Médecine; and H. FRUCHAND, Membre Correspondant de la Société de Chirurgie. Pp. 600, with 202 illustrations and 32 radiograms. Price 120 francs. Gaston Doin et Cie, Paris, 1935.

The authors are to be congratulated on a work which gives all the latest knowledge of chest surgery in one volume. It contains details of the various surgical procedures, with indications for their employment, and full reference to other authorities. Apicolysis with paraffin wax is an operation which attracts many followers, not only because in theory it is an ideal method of collapsing an apical cavity, but because it is safer, less disfiguring, and less drastic than thoracoplasty. In practice, however, the operation has not given the results expected of it. The authors devote forty-six pages to an impartial discussion of this operation and its results.

One chapter deals with anæsthesia, and the pros and cons of local or general anæsthetics are discussed. Post-operative complications and their prevention and treatment form the subject of another chapter.

The book is well produced and illustrated, and should prove a valuable addition to the library of the thoracic surgeon.

Tuberculosis of the Lymphatic System. By RICHARD H. MILLER, M.D., F.A.C.S., Assistant Professor of Surgery Harvard Medical School. Pp. 248. Price 17s. Macmillan and Co., New York, 1934.

This is a practical book by an author who has gained his experience at the bedside rather than in the laboratory. Descriptions of tuberculosis of the cervical, tracheo-bronchial, abdominal axillary, inguinal, and other groups of glands will be found, with full details of treatment. In the early chapters the anatomy and biology of the lymphatic system are described, and one chapter is devoted to modes of entrance and spread of disease. References are given at the end of each chapter, and there are many excellent illustrations.

Tuberculosis of the lymph glands is rapidly becoming less common, and the author thinks is it gradually disappearing in the United States of America. The recognition of the lymphatic system as a protection and as a channel of spread is of the utmost importance, and this work was much needed and forms a welcome addition to the literature of tuberculosis.

Endoscopie et Pleurolyse. By O. M. MISTAL, Médecin Directeur de l'Etablissement Clovelly, Montana, Switzerland. Pp. 436. Price 55 francs. Masson et Cie, Paris, 1935.

This book, which has a preface by Professor Jacobæus, is a veritable treatise on the methods of examining the pleural cavity with a thoracoscope and the cauterisation of adhesions. A description of the various adhesions are given, together with their microscopic appearances. A long list of the instruments used and their description, together with the technique of different operators, will be found. No detail has been omitted, and at the end of the book references to 557 authors will be found. There are numerous skiagrams and illustrations, and a full index which is well arranged.